

Pattern Hair Loss: Genetics, Pathophysiological Mechanisms, Dermatological Assessment, and Treatment Approaches

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ABSTRACT

Pattern hair loss (PHL) occurs in both men (MPHL) and women (FPHL). Some studies estimate that about 50% of males will develop PHL by the time they turn fifty, and that percentage increases to over 70% by the time they turn eighty. The original purpose of minoxidil was to treat hypertension. This treatment mentioned hypertrichosis as a side effect, and this hair loss application was developed. The only medication known to work for both men and women is topical minoxidil, but it remains uncertain how it actually works. Twenty or so years ago, there wasn't any certain treatment or cure for balding in men or women. The pathophysiology of this common disorder was unknown till 1940. Hair cycle has a few phases, e.g. anagen, catagen, telogen, early to mid-anagen (regrowing phase) and again anagen (new growing phase). In other words, there is a "final common pathway" of follicular miniaturization, meaning that the follicular alterations in MPHL and FPHL appear to be the same regardless of the origin. This entails a notable reduction in anagen length. Dermatological Assessment includes hair thinning, hair fall tests, and hair pull tests. Treatments include the use of minoxidil and surgical treatment. The genes involved in hair fall are Wnt/ β -catenin signaling pathway, EDA2R on the X chromosome.

Keywords: Pattern Hair Loss (PHL), Genetic Hair Loss, Male Pattern Hair Loss (MPHL), Female Pattern Hair Loss (FPHL), Wnt/ β -Catenin Pathway Genetics, Minoxidil, Anagen Duration, Hair Regrowth, Hair Loss Pathophysiology, Dermatological Assessment, Treatment for Hair Loss, Hair Follicle, Hypertrichosis Side Effect

INTRODUCTION

Humans are naturally predisposed to have thick, long hair due to about 100,000 hair follicles. According to Paus [1], hair goes through three separate and simultaneous phases in a precise, recurring development cycle: telogen, which occurs three to four months prior to hair shedding; catagen, which lasts two to three weeks; and anagen, which lasts three to five years. One hundred hairs are usually lost from the head per day [2]. Nonetheless, a higher incidence of physiological deterioration is a worldwide issue. Hair releases and sheds during the period of rest or telogen [3], initiating the next cycle at any given time. About half of women have visible hair loss by the time they are sixty years old, although men often experience hair loss by the age of fifty.

Both men (MPHL) and women (FPHL) are affected by pattern hair loss (PHL). According to some studies, by the time they turn fifty, over 50% of men will have PHL, and by the time they turn eighty, that number rises to over 70% of males [4]. Pattern hair loss affects 3–13% of women under 40 and 32–54% of women over 70, which is far less common in women than in males. There are still many unanswered questions regarding the complicated pathophysiology of hair loss, particularly in the case of FPHL. Before going into a period of rest, the human scalp goes through up to six years of anagen (growth), during which the hair shaft develops and grows. [3] and the release of the hair shaft (exogen). While we do not yet understand the signals responsible for the switch between the various hair cycle phases, we believe that complex signaling cycles are important between the dermal papillae (DP) and the root sheath, involving multiple families of proteins including fibroblast growth factors (FGFs), bone morphogenic proteins, sonic hedgehog and WNT signaling. Normally, approximately 90% of follicles are in the anagen phase of hair cycling,

about 9% of follicles are in the telogen phase and 1% are in the catagen phase. The proportion of follicles in anagen decreases with age. In PHL, the hair cycling appears to be dysregulated and out of balance, with a shortened anagen stage, increased proportion of hair in catagen/telogen stage and a change in hair type to shorter, finer hairs. The estimated percentage of follicles in the hair cycle's phases are: Anagen – 90%, Catagen – 1%, Telogen – 9%. With age, the percentage of follicles in anagen decreases. Pattern hair loss is defined by 7 problems of hair cycling; a shortened anagen, increased percentage of hair in catagen/telogen, and a shift towards making fine short hair. There are still many unanswered questions regarding PHL, including the mechanisms underlying the unbalanced hair cycle stages and follicle shrinking. Many cases in males involve androgens, leading to the general application of the umbrella term of androgenic alopecia to PHL[5]. However, the aetiology of pattern hair loss, or pattern baldness, remains poorly understood, especially in women. The low response rates to many of the current treatments may be a reflection of this condition. However, it is well recognized that dihydrotestosterone (DHT), which is produced from testosterone by 5 α reductases, is important in many men. Although the intracellular signalling cascade following DHT's binding to androgen receptors remains unclear, the binding of these receptors triggers the creation of transforming growth factor and other cytokine factors 1 (TGF1), which aids in telogen and DP cell senescence.

In females, it's still unknown how androgens play a role. The aetiology of FPHL has been connected to hormonal changes in postmenopausal females, although a life-course decline in hair density that only manifests later in life could possibly be the cause of

these changes. However, the fundamental processes of FPHL are still unknown. The Food and the FDA has given finasteride approval and minoxidil for the treatment of hair loss, when used alone or together. MPHL treatment with 5α reductase inhibitor (5α -RI) has been explored with the idea that it will block the conversion of testosterone to DHT, which is beneficial. One of the many negative side effects of finasteride is sexual dysfunction, which can sometimes persist long

after you've finished taking the drug. Because only off-label, uncontrolled research and anecdotal evidence have shown good effects, the use of finasteride in women is contentious despite the small number of studies that support its benefits. 13. Because finasteride is a teratogenic substance, treating it in premenopausal women can potentially be troublesome and necessitates concurrent contraceptive medication.

Hair Growth Cycle

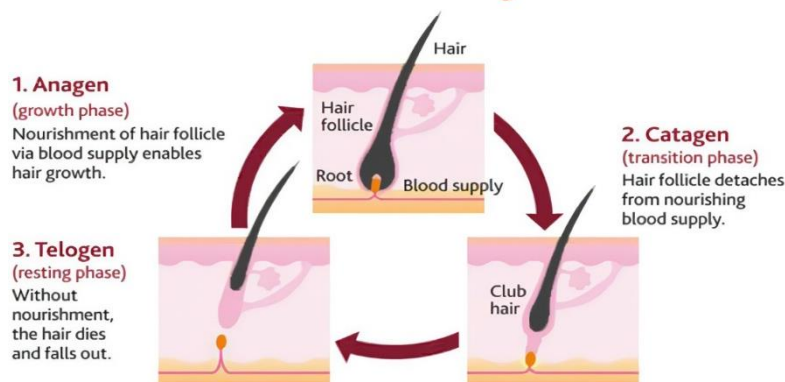


FIGURE 1: Hair Growth Cycle

RESEARCH AND REVIEW

Initially, researchers developed Minoxidil to treat hypertension [6]. The treatment noted hypertrichosis as a side effect, leading to the repurposing of the product as a treatment for hair loss. The only medication that works for both sexes is topical minoxidil, although its specific mechanism of action is unknown. Despite being widely recognized, minoxidil can have several side effects, such as initially increasing hair loss and making it worse after discontinuing the medicine [7]. FGF5 (fibroblast growth factor 5) was found to be a predictor of the length of the hairs in groundbreaking research using FGF5 knock-out mice, which exhibited the angora long-haired phenotype and a longer anagen phase [8]. 18 Researchers discovered that familial trichomegaly is caused by mutations in FGF5, or Growth factor 5 for fibroblasts, which is a key regulator of human hair length. Compared to ethnically matched controls, People that carried this mutation (FGF5) had longer hair and more hair follicles during the anagen stage. Research on rodents has revealed an upregulation of

FGF5 messenger RNA in the late anagen period, the responsiveness of DP cells expressing the FGF5 receptor (FGFR1) to fibroblast growth factor 5 in vitro²², and the inhibition of DP cell activation by FGF5. 23 such results show that a crucial regulating mechanism is fibroblast growth factor 5 signalling to DP cells via FGFR1. Additionally, fibroblast growth factor 5 is expressed more frequently outside the root sheath and in the microscopic cells that surround the follicle and resemble macrophages at the conclusion of the anagen. When FGF5's messenger RNA is spliced differently, it makes the long (FGF5) and short (FGF5S) isoforms, which work together. There is a chance that outside substances that block fibroblast growth factor 5 could help hair grow because they work against the effect of FGF5 on catagen. We looked at plant extracts for their ability to stop fibroblast growth factor 5 from working because there is evidence that it slows hair growth and plays a part in the change from anagen to catagen. We found that *Sanguisorba officinalis* root extract (SO extract) effectively blocked fibroblast growth factor 5 and showed that SO extract increased the growth outside the root sheath in the lab [5].



FIGURE 2: Minoxidil Structure

Twenty years or more ago, there was no particular remedy for the hair loss in both sexes. It wasn't until 1940 that the aetiology of this prevalent condition was understood. FPHL is now treated with topical minoxidil, spironolactone and cyproterone acetate. Spirolactone and cyproterone acetate are two antiandrogens which inhibit high affinity androgen receptors. They have similar molecular structure consisting of a pentane ring and a steroid structure consisting of three hexanes. Due to its acetylthio substitution at position 7, spironolactone, an aldosterone antagonist, acts mainly as a potassium sparing diuretic and antihypertensive agent. It also acts as an antiandrogen, competes with androgen for binding to androgen receptors and suppresses the production of androgens by the ovary; at doses of 50-300 mg/day it has

proven effective for the treatment of hirsutism and acne. This medicine has some side effects such as postural hypotension, electrolyte imbalance, renal insufficiency, irregular menstruation, fatigue, and urticaria. The androgen receptor blocker cyproterone acetate has a slightly reduced glucocorticoid action and high progestational activity. Hair shedding is reduced in FPHL, but no discernible regrowth occurs. However, cyproterone acetate has not received approval from the US Food and Drug Administration. For ten days during each menstrual cycle, premenopausal women need to take approximately 100 mg each day. Every day, postmenopausal women take 50 mg. Side effects include sadness, libido loss, breast soreness, weight gain, and irregular menstruation.

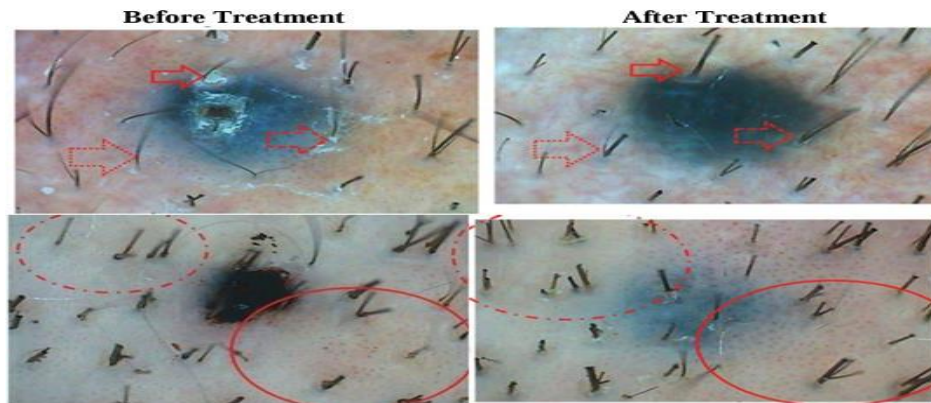


Figure 3: At Week 24, the Average Hair Growth of Two Participants had Improved Moderately (Before, After)

The severity of MPHL grows with age, and when they reach the age of 70, at least 80% of Caucasian males exhibit some symptoms of the condition. It's unclear if thinning after the ages of 50 to 60 is solely androgen-related or if it's a side effect of another ageing-related element. Compared to Caucasians, Asian, American, and many African-American men experience less frequent and less severe frontal hair loss. Androgen is responsible for male pattern hair loss. Among Hamilton's study, MPHL was not present among men who had been castrated before puberty. Nevertheless, MPHL was observed in 4 out of 12 male castrates that received testosterone treatment. When complete androgen insensitivity syndrome is present, the androgen receptor expression is completely absent, MPHL has not been documented [9]. Despite being the main androgen in men's blood, dihydrotestosterone, a byproduct of testosterone, is essential for MPHL. 5 alpha R catalysis the conversion of testosterone to dihydrotestosterone. Five alpha R has two isoforms, each of which is expressed by a separate gene and has a different tissue distribution. Despite being abundantly expressed, type I 5 alpha R's physiological role is unclear. Two examples of androgen-

dependent tissues that express type II 5 alpha R are the prostate and hair follicles. Men with a hereditary Type II 5 alpha R deficiency do not have MPHL. After taking finasteride, almost two-thirds of men report having more hair, which inhibits the progression of MPHL and is a specific inhibitor of Type II 5 alpha R. Although it is unclear what the primary target of androgen action is, there is evidence that the tissue (dermal papilla [DP] and dermal sheath [DS]) is derived from mesenchyme. Although it is unknown what these variables are and how they are inherited, MPHL is predisposed by hereditary factors. MPHL is polygenic in its inheritance. Sons of males with MPHL have a higher incidence of hair loss in men. The effect of moms on MPHL is not well understood. MPHL's early onset could indicate if a woman is a carrier of a gene that causes polycystic ovarian syndrome [10]. The gene's identity is unclear. MPHL, especially early onset, is associated with polymorphism in the gene for the androgen receptor. However, the paternal influence in males with MPHL cannot be explained by the androgen receptor gene, which is located on the X chromosome and is therefore exclusively inherited from the mother [11].



Figure 4: Androgenetic Alopecia in the Hair Loss Pattern in both Sexes

In Women

A "male" hair loss pattern is displayed by these hyperandrogenic women. The authors proposed using the broader term "female hair loss pattern" instead of "androgenetic alopecia" because it is unclear how androgens play a part in all cases of FPHL [12]. Without a doubt, women with hyperandrogenism experience FPHL. A "male" hair loss pattern is displayed by these hyperandrogenic women. Other hyperandrogenism symptoms, such as hirsutism and/or irregular menstruation, are present in these women. Although hyperandrogenism does not necessitate a complete increase in serum androgen levels, it does imply a greater expression of androgen-related disorders. Finasteride or cyproterone acetate are usually effective treatments for women who have pattern hair loss with hyperandrogenism. However, as can be seen from the clinical findings below, women with FPHL typically experience a distinct pattern of hair loss from males with MPHL [13]. According to Carmina et al. [9], FPHL women typically exhibit no additional clinical or biochemical signs of androgen excess. However, FPHL women have more

complicated family histories than men with MPHL. Additionally, neither women without indications of androgen excess nor postmenopausal women responded to finasteride therapy or cyproterone acetate.

GENERAL

MPHL and FPHL seem to have the similar follicular changes, regardless of the origin; in other words, there is a "final common pathway" of follicular shrinkage. This entails a notable reduction in anagen length. Although this has not yet been confirmed in females, the latent duration of the hair cycle has been prolonged. After a telogen phase, during which the hair is shed, anagen typically reasserts itself. Additionally, following the hair shaft's dropping, the growth of the follicle is persistently suspended during the latent phase [14].

Hormonal Impact on Hair at Various Phases of the Lives of Women

Based on a woman's life stage, hormones affect her hair (Table 1).

Table 1: The Impact of the Hair Cycle's Hormones across Various Stages of a Woman's Life

Life Stage	Key Hormones Activated	Impact on Hair
Puberty	Androgens	The pubic and axillary regions transform vellus hair into terminal hair.
Androgen Excess	(e.g., PCOS, NCAH, Cushing's syndrome, hyperprolactinemia)	Hirsutism (excessive body hair growth).
Reproductive Age	The function of sex hormones is unclear or inexplicable.	Female pattern: hair loss.
		Hyperthyroidism (thyroid imbalance-related hair thinning). Alopecia areata (immune-related hair loss). Hypothyroidism (hypothyroidism-related diffuse alopecia).
Pregnancy	Growth factors, prolactin, progesterone, and estrogen are all at higher levels.	There has been an increase in hair growth, a thicker hair diameter, and a higher anagen/telogen ratio, which indicates a longer hair growth phase.
Puerperium	Reduction in progesterone and estrogen	The temporary hair shedding that occurs after childbirth is known as postpartum telogen effluvium.
Menopause	Estrogen depletion	Women experience hair loss and hair thinning. Facial hirsutism (excessive facial hair growth).

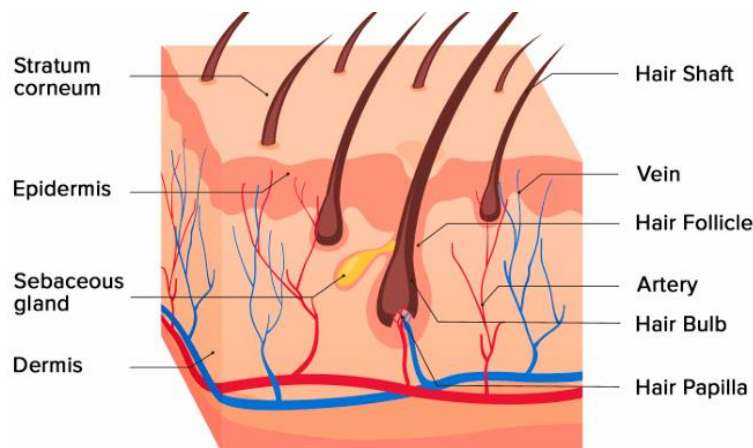


Figure 5: The Shaft and Hair Follicle Architecture are Depicted in this Figure

Table 2: The Impact of the Hair Cycle's Hormones across Various Male Life Stages

Life Stage	Key Hormones Activated	Impact on Hair
Puberty	Androgens (testosterone, DHT)	The face, chest, and underarms are where Terminal hair develops from vellus hair. Thicker hair on the scalp.
Androgen Excess	Elevated Testosterone and DHT	Male pattern baldness (androgenetic alopecia) typically begins, often first appearing at the temples and crown. Increased body and facial hair.
Young adulthood (late teens to early 30s)	Testosterone, DHT	Healthy, thick scalp hair. Hair grows on the face, chest, and body. Male pattern baldness may begin in genetically predisposed men.
Mid-adulthood (30s to 50s)	Declining testosterone, stable DHT levels	There is a noticeable thinning of scalp hair, particularly in areas sensitive to DHT such as the temples and the crown. Progression of male pattern baldness. Reduced body hair growth.
Late Adulthood (50s and beyond)	There is a decrease in testosterone and alterations in other hormone levels, such as estrogen, among older men.	The hair thinning continues, particularly on the scalp. Male pattern baldness may worsen. Reduced facial and body hair growth.

The study examined hair loss in both genders. Somia Gul, Tahira Khan, Shagufta Naz, Abeeha Malick and Sameera Kaleem [15] conducted a survey of 200 people (n=200) who suffered from hair fall and were collected from different areas in Karachi, Pakistan. They analyzed all the volunteers, ranging in age from 18 to 40. These are the primary causes of hair breakage and ultimately hair loss, so they took a look at a number of factors including genetics, anaemia, environment and overuse of chemicals on the hair. The hair fall products that their volunteers frequently used for hair fall therapy were examined. Plotting graphs of the variables influencing Male Pattern of Hair Loss (MPHL) and Female Pattern of Hair Loss (FPHL), various

age groups with alopecia, and the various products used by volunteers allowed them to statistically assess their findings [15].

Results of Analysis

28% of the participants used solution sprays for hair fall, such as Minoxidil, Romaine, Rogaine, and Doctor; of these, Romaine and Doctor were effective for a specific period, after which they increased hair fall. Conversely, minoxidil demonstrated its effectiveness even after prolonged use and yielded positive results. Several other studies have been conducted in various parts of the world, and the following are the results:

Table 3: Results of Several other Studies Conducted in Various Parts of the World

Category	Details	Percentage Affected	Source Output:
Gender Distribution	Men	80% by age 70	Verywell Health, Mayo Clinic
	Women	38% by age 70	Verywell Health, Mayo Clinic
Contributing Factors	Genetics (Male Pattern Baldness)	95% of male hair loss cases	Medihair, JAMA Dermatology
Common Treatments	Alopecia Areata	0.2% of the population	JAMA Dermatology
	Minoxidil (Topical)	FDA-approved for hair loss	Mayo Clinic
	Finasteride (Oral)	FDA-approved for male pattern baldness	Mayo Clinic
	Hair Transplant Surgery	Increasingly popular	The Sun, Medihair
Age Group Prevalence	Under 20	20% of men experience hair loss.	Medihair, Verywell Health
	By age 40	80% of men experience noticeable hair loss.	Verywell Health, Medihair

DERMATOLOGICAL ASSESSMENT

Hair Comb Test

Day 60 and Day 90 dermatological assessment results showed significantly less hair fall with and without bulbs compared to baseline. After the research, hair fall with bulbs decreased by 57.53%, and hair fall without bulbs decreased by 81.60% (Figure 6).

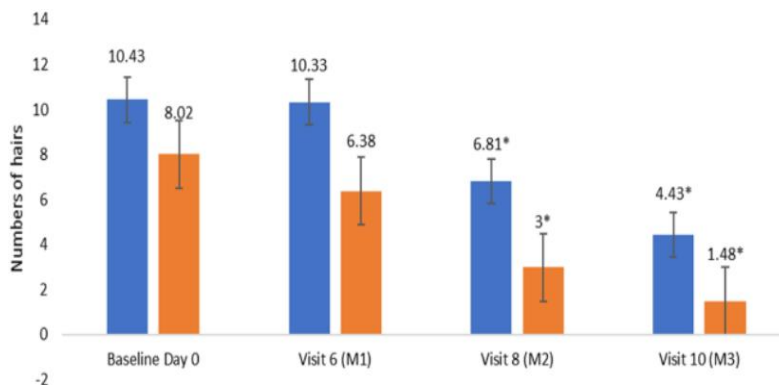


Figure 6: Comb Test Dermatological Evaluation. We Talled the Quantity of Hairs Obtained Following the Hair Comb Test at Visits 1, 6, 8, and 10 and categorized them as Hair Falls both with and without a Bulb. The Mean $\pm$ SE; *P is Used to Express Values

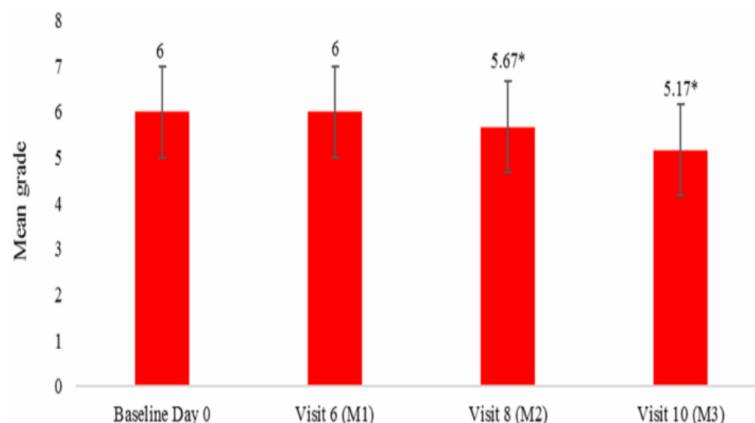


FIGURE 7: Evaluation of Hair Thinning

The evaluation of hair thinning was conducted by comparing images. The MSCR 10-point photo-numeric hair thinning scale was used to evaluate hair thinning, and the results were compared to images from VISIA CR imaging. A better outcome is indicated by a lower grade. Mean $\pm$ SE; *p is used to express values.

Hair Fall Test

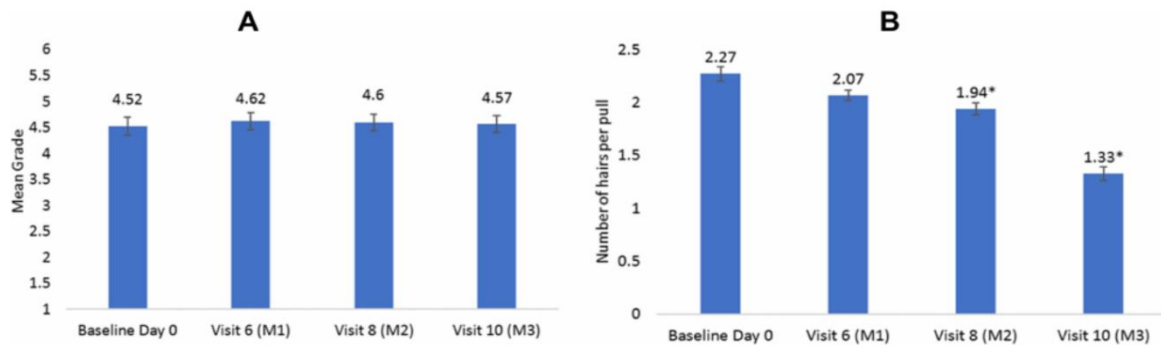
There was no discernible change between hair fall at any time point and baseline for the linear photometric scale (Figure A). There was no change in the grading for thirty-eight subjects, and three topics'

grades improved from 3 to 4, while one subject's grade went from 6 to 4.

Test of Hair Pulling

The hair pull test is used to evaluate the tightness of the hair papilla anchoring [16].

By the end of the research, the average number of hairs taken each draw had dropped from 2.27 to 1.33 (a reduction of 41.4%) from the baseline. As early as day 30, we noticed a statistically significant shift (Figure B).



FIGURES 8 A AND B: (A) To measure hair fall, the linear photo-numeric scale was employed. During research visits, we directly compared the individuals' scalps to the MSCR 10-point photo-numeric hair thinning scale to assess hair loss. A better outcome is indicated by a lower value. Mean grade $\pm$ SE is used to express values. (B) Evaluation findings for the hair pull test. As the fingers traced the hair shaft, we forcefully pulled hair from the scalp and counted how many hairs were removed. Values are indicated by the mean number of hairs per pull $\pm$ SE; *p.

TrichoScan Hair Density Assessment Instrument

As early as day 30 and throughout all time points, the test product's mean hair density rate showed a significant improvement

over baseline, according to TrichoScan's statistical analysis. (Figure 9A). The percentage change was 7.97%, as the density rose from 171.35 ± 21.9 hairs/cm² to 185.01 ± 20.57 hairs/cm² [17].

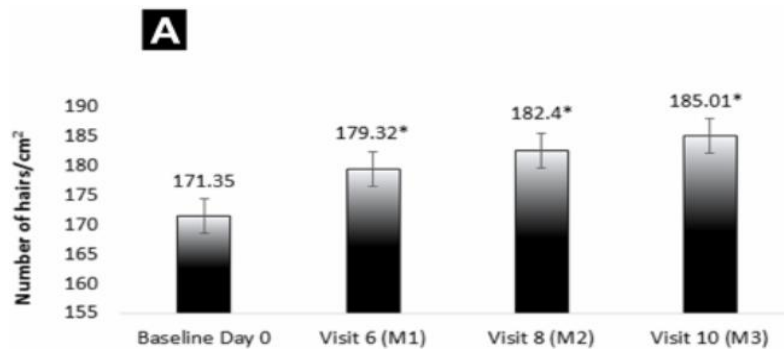


FIGURE 9 A: Assessment of Hair Fall

Rate of Hair Growth via TrichoScan

When compared to baseline, the test product's continuous application resulted in notable increases in hair growth rate on Days 30, 60, and 90. The average growth rate increased by 17.36%, from $306.39 \mu\text{m}$ to $359.56 \mu\text{m}$ every two days (Figure 9B) [18].

TrichoScan's Anagen and Telogen %

Over the course of the trial, the proportion of anagen and telogen hairs was constant.

The mean percentage of telogen or anagen hairs did not decrease (Figure 9C).

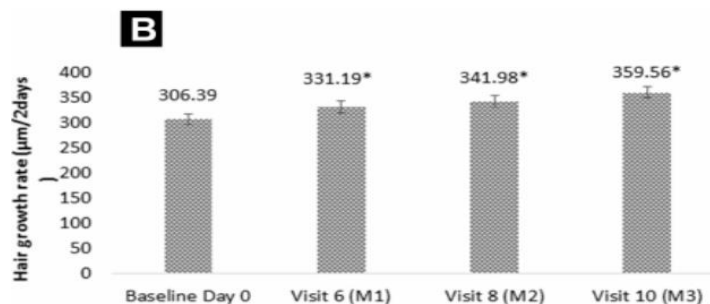


FIGURE 9 B: Assessment of Hair Growth

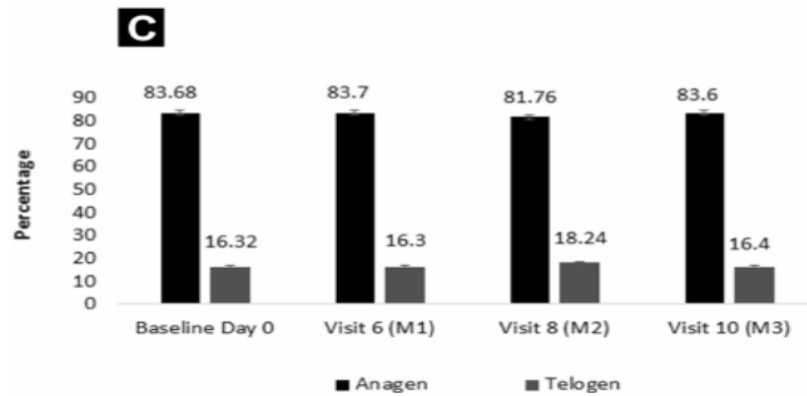


FIGURE 9 C: Proportion of Anagen and Telogen Hairs

Density of Vellus and Terminal: TrichoScan

The density of the terminal hair and vellus was assessed using TrichoScan software. At every time point, the terminal hair density

increased over baseline in a statistically meaningful way (Figure 9D). At Visit 10, the percentage change in terminal hair density and vellus density relative to baseline was 5.93% and 13.51%, respectively.

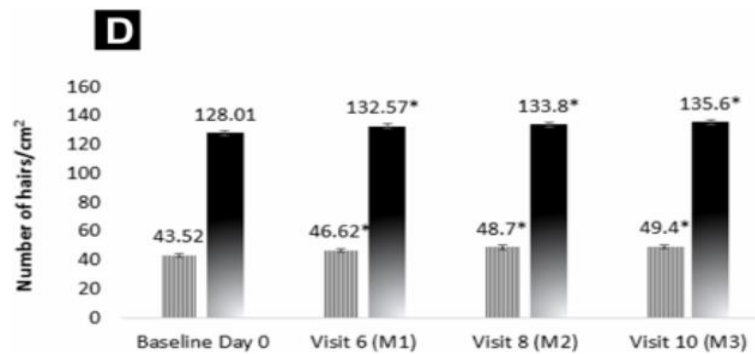


FIGURE 9 D: No of Hair

The TrichoScan evaluation of a 1 cm shaved section of the scalp is shown in Figures a, b, c, and d. The number of hairs per centimeter², or hair density, is measured. In Panel (A). As a representation of the hair growth rate assessment in Panel (B), the length of hair grown every two days is shown. The percentages of telogen and anagen hairs are shown in Panel (C). Panel (D) shows the number of hairs per centimeter (vellus and terminal hairs). We express the values as mean, SE, and *p.

Hair Fall Types

Alopecia androgenetica

Men and women who are genetically predisposed to androgenetic alopecia may experience it at any stage of life, from puberty to old age. The patient's main complaint is thinning; however, the initial occurrence is primarily telogen shedding [19].



FIGURE 10: Alopecia Pattern

Reasons

Hair loss is stimulated by androgens such as dehydrotestosterone and dehydroepiandrosterone sulfate.

A powerful androgen, dehydrotestosterone, increases body and genital hair while decreasing scalp hair.

Androgenetic alopecia affects both sexes equally.

Presenting Alopecia Areata

Many patches, total loss of scalp hair (alopecia totalis), and whole scalp and body hair loss are the clinical manifestations of alopecia areata, which cause single or recurrent patchy hair loss [20].

Reasons

The shedding phase is accelerated in alopecia areata due to a profound inflammatory process surrounding the follicle.

While the irritation persists, the affected hair falls out. And no new growth appears.

There is currently no known aetiology for alopecia areata.



FIGURE 11: Alopecia Areata, Both Men and Women Patterns

The Telogen Effluvium

One of the most usual types of hair loss is known as telogen effluvium. In the disease, the hair follicles enter the resting phase, or telogen phase, too soon from the growth phase, according to Grover and Khurana [21].

Reasons

Three to six months before the start of the shedding, an event sets off the process. At first, acute shedding was linked to several circumstances, including feverish illnesses, delivery, chronic systemic diseases, heparin use, and mental distress.



FIGURE 12: Telogen Effluvium, Both Male and Female Patterns

Alopecia Traction

Although traction alopecia is chronic and recurrent, it might alter the hair growth cycle and cause physical damage to the hair shaft. In the frontal region of the head, traction alopecia results in sparse and broken hair [22].

Reasons

Nearly all cases are caused by rigid braids, elastic hair bands, ponytails, rollers, and other hairstyles that significantly and continuously stress the scalp hair.



FIGURE 13: Male and Female Patterns of Cicatricial Alopecia

Diffuse Hair Loss and the Hair Cycle

Scalp hair grows in four cycles: anagen (growth), catagen (involution), telogen (resting), and exogen (release of dead hair). Diffuse loss can result from a variety of causes that interfere with the

regular cycle of hair development. The most prevalent kind of diffuse shedding is caused by the diffuse failure of telogen-phase hairs.

Usually, chemotherapy and radiation treatments are the cause of diffuse hair loss during the anagen period [23].

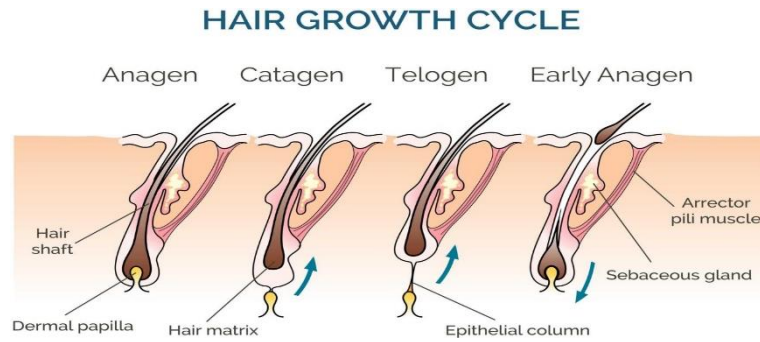


FIGURE 14: Hair Cycle

Dandruff

A cluster of corneocytes known as a "dandruff scale" is formed when they separate from the stratum corneum's surface while maintaining a high degree of cohesiveness among themselves. It might have a pilosebaceous follicle at its centre. The scales vary in size and quantity depending on where on the scalp they are found. A

portion of dandruff is composed of parakeratotic cells. The number of clinical symptoms is associated with their intensity, which can be affected by seborrhea. Thus, pityriasis simplex, sometimes known as "sicca," denotes the presence of tiny, generally asteatotic squames that give the process a farinaceous appearance. In contrast to steatoid pityriasis or "oleosa," steatoid dandruff is seborrheic, thick, and sticky, and its scale is frequently greater [24].



FIGURE 15: Dandruff in Hair

TREATMENTS AND EFFECTIVENESS

Surgical Treatment

The most successful treatment for this issue is still hair transplants, which use hair grafts. It successfully replaces bald spots with hair that looks nearly 100% natural. In their outpatient clinics,

dermatologists and plastic surgeons in North America provided care for both sexes. Under local anaesthesia, it is performed. The donor strip is taken out of the skull's back. Microscopic tools will be used to cut the strip into smaller pieces. We naturally place tiny grafts into the recipient region. Patients require "1 to 6" methods at intervals of "6 to 12" months. Each session can implant between 1,000 and 3,000 hairs [25].



FIGURE 16: Surgical Treatment

Alopecia Areata

The three primary forms of alopecia areata are baldness of the entire scalp (alopecia totalis), baldness of the entire body, and localised bald spots in the scalp, brows, or beard areas. Hair loss outputs from inflammation of the hair roots caused by an immune

system malfunction. When the hair starts to become white, the situation can get worse. This illness is greatly influenced by changes in the mind and emotions. This disease is frequently confused with an infection. Exclamation-mark hair and tapering hair follicles are present at the proximal end, although there are no indications of

scarring. Numerous other immunological disorders, including vitiligo, thyroid, and pancreatic disorders, may be linked to this

syndrome. In order to rule out the aforementioned disorders, the specialist must perform all required blood tests [20].



FIGURE 17: Alopecia Areata

We treat alopecia areata based on the patient's age. Topical medications treat children under 10 years of age. This includes short-term contact thralin therapy, a 5% minoxidil solution used as a hair tonic, and the use of potent steroids (such as lotions or gels). The percentage of the damaged areas determines the therapy for adults, children, and those over ten. When 50% or less of the scalp is affected, strong steroids (creams or skin injections), minoxidil 5%, anthralin, or any combination of these might be suggested.

The best course of treatment is DPCP (diphenylcyclopropenone) if 50% or more of the scalp is affected. It prevents the cutaneous immune system from overreacting. Many people found hope in this treatment. Its use should be supervised by the medical staff. Anthralin and photochemotherapy (PUVA) are further treatments. Other therapeutic options include cyclosporine and corticosteroids. We do not recommend that patients continue taking them because of the long-term adverse effects. In the event that none of the previously

mentioned solutions work, wearing a cosmetic hair wig will be the last resort.

Trichotillomania

An insatiable need to pull out one's hair is a defining feature of trichotillomania, a mental illness that results in non-scarring alopecia. The ratio of females to men in this scenario is 3.5:1. On the opposite side of the right or left-handed person, the disorder presents as non-scarring alopecia patches with hair follicles of different lengths. This is a disorder known as impulse control disorder. Competitive presentations and the fact that most patients do not wish to accept the prospect of hair pulling means that alopecia areata and other non-scarring causes of alopecia should be ruled out. After confirming the diagnosis of trichotillomania, a psychiatrist should be consulted for treatment. A psychiatrist can provide different therapeutic options such as behaviour therapy, hypnotic therapy, psychotherapy and pharmacotherapy [26].



FIGURE 19: Trichotillomania

Tinea Capitis

Capitis means the scalp and tinea is a fungal disease. In the past people used to say "ringworm". The lesions were arranged in rings, hence its name. Dermatophytes are a common cause of tinea capitis, and often it is the root cause of children's hair loss. Dermatophytes are categorized as favus, endothrix, or ectothrix when they infiltrate the hair. Favus is an inflammatory condition with hairy, yellow-crusted plaques that is brought on by the fungus *Trichophyton schoenleinii*. If the fungus grows inside the hair shaft, endothrix is formed, mostly by *Trichophyton tonsurans*, *Trichophyton soudanense* and

Trichophyton violaceum. Hyphae are found both within the hair shaft and outside the hair shaft of ectothrix which include *Trichophyton verrucosum*, *Microsporum audouinii*, *Microsporum canis*, *Microsporum distortum*, *Microsporum ferrugineum*, *Microsporum gypseum* and *Microsporum nanum*. The types of fungus that cause the disease can occasionally be different within a geographic area and from one geographic region to another. In the US and Europe, *T. tonsurans* is the main cause of tinea capitis (replacing *M. audouinii* and *M. canis*). Although tinea capitis is mainly found in children, adults can also get it. If young people share their hats, combs and hairbrushes they could also become infected. Tinea capitis may occur

in different clinical patterns such as inflammatory or "kerion", favus, "black dot" (like seborrheic dermatitis) and alopecia areata. All types of tinea capitis can be associated with the occurrence of cervical or occipital lymphadenopathy. Untreated tinea capitis infections may result in scarring alopecia. The fungal hyphae and spores can be found by microscopic analysis of the broken hairs using 10-15% KOH on a slide. Fungal cultures can be best obtained by gathering the scales infected with the fungus by rubbing a sterile toothbrush against a scaly scalp. For children it is recommended to use griseofulvin, "20-25 mg/kg/day approximately dose.

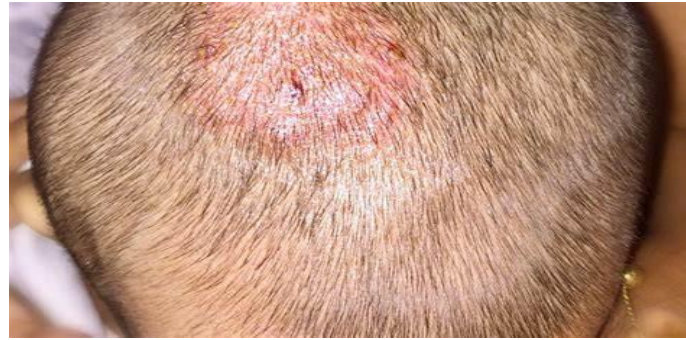


Figure 19: Tinea Capitis

Genetic Involvement

The gene for the androgen receptor (AR) is most strongly linked to pattern hair loss; the gene makes the follicles more sensitive to the effects of DHT and makes the process of miniaturization happen more quickly. There's a big genetic component to this. Other important genes involved include those in the Wnt/ β -catenin signaling pathway which is required for the growth and cycling of hair follicles, and other genes such as EDA2R on the X chromosome that have a strong association to hereditary baldness. Polymorphisms in the gene coding for 5-alpha reductase (SRD5A2) also can enhance the production of DHT, while genes coding for prostaglandins and prostaglandin metabolism (such as PTGDS for prostaglandin D2 synthase) can add to the inhibition of hair production. These genetic factors together create an inclination which determines the time at which hair loss begins, the rate of hair loss and the response to a therapy.

Discussion

Rogaine, Romaine, and Doctor are among the hair fall solution sprays used by 30% of the population. After a certain amount of time, hair fall solutions start to increase hair loss. Minoxidil, however, works even when left untreated and produces favorable results. Arnica, coconut oil, mustard seed oil, and jojoba oil are among the herbal products used by 22%. There are exactly 200 individuals in the population for this survey, including both all ages of males and females. The majority of young participants between the ages of 18 and 30 had alopecia. According to the results of our survey, 68% of men had hereditary baldness and 60% of women experienced hair loss problems due to inheritance, making inheritance the most prevalent cause of male baldness. Only 15% of male volunteers were anaemic, compared to 26% of female volunteers. The main issue facing women nowadays is anaemia, which is caused by poor iron consumption. According to our survey results, poor intake of iron and protein, environmental variables like stress and salty water, excessive chemical treatment, and hair styling techniques like dyeing and straightening are other reasons that may contribute to hair fall. It is "surprising"

Overall, it is fairly well tolerated and safe drug. This is recommended to be approximately "6 to 8" weeks. A wonderful alternative is "Terbinafine." It is a medication that is taken by mouth in doses of 62.5mg/day for "11-20kg" of body weight or 125mg/day for "21-40kg" or 250mg/day for "over 40kg" for "2-4" weeks. It's a fungicidal substance. The most common side effects are skin rashes and stomach problems. A novel alternative antifungal medication is itraconazole. For four to six weeks, a dose of about 5 mg/kg/d is given. The medication is comparatively safe. The most common adverse effect is gastrointestinal distress [27].

how little we know about the causes, incidence, prevalence, and treatment options for hair loss. The survey form was based on a supplementary questionnaire of the scalp, family history, illnesses and other factors that could have an impact on hair development.

In females, anaemia or iron deficiency in the blood is the primary reason for hair loss. This results in pallor of the body and the follicles becoming weaker and causes hair loss. Patients should consume foods that are high in iron such as fish, dried fruits, legumes, pork, potatoes, molasses, dark green leafy vegetables, wheat germ, and grain products which will help prevent hair loss. It is significant to note, however, that excess vitamin A could cause hair loss. Caused by genetic changes, earliness of hair loss in boys was mostly inherited and could be due to either the mother or the father's pattern. The hormone testosterone's conversion to dihydrotestosterone is the main factor causing hair loss. Hair loss has been treated using rogaïne in cases of hereditary hair loss, because the follicles are smaller, thereby limiting hair development. Many people treat their hair loss by themselves but aren't sure about the exact length, duration and treatment method. They overapply the product because they are unaware that every person has a particular hair type and that not all products are good for their hair. For as long as they like, these patients can continue or stop the treatment. The patient should speak with their doctor since the treatment should be stopped and an alternative therapy should be utilized if they do not show any signs of recovery after four to six months. Both men and women take minoxidil 2% because it encourages the growth of facial hair, while minoxidil 5% * 10% is not advised for women. The minoxidil is absorbed by the skin, increasing blood supply to the follicles and so maintaining hair growth.

Conclusion

In conclusion, both sexes are affected by pattern hair loss (PHL). According to some studies, by the time they turn fifty, roughly 50% of males will have PHL, and by the time they turn eighty, that number rises to over 70%. Initially, researchers developed Minoxidil to treat hypertension. After hypertrichosis was discovered as a side effect, the

drug was repurposed as a treatment for hair loss. The only medication that is known to work for both men and women is topical minoxidil, albeit its exact route of action is uncertain. There was no particular remedy or cure for hair loss in either men or women a few decades ago. The pathophysiology of this common disorder was unknown till 1940. Hair cycle has a few phases, e.g. anagen, catagen, telogen, early to mid-anagen (regrowing phase) and again anagen (new growing phase).

In other words, there is a "final common pathway" of follicular miniaturization, meaning that the follicular alterations in MPH and FPHL appear to be the same regardless of the origin. This entails a notable reduction in anagen length. Dermatological Assessment includes hair thinning, hair fall tests, and hair pull tests. Treatments include the use of minoxidil and surgical treatment. The genes involved in hair fall are Wnt/ β -catenin signaling pathway, EDA2R on the X chromosome.

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