Review Article

Alopecia areata: Pathophysiology, causes, and herbal treatment

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How to cite this article: Kaur J, Kaur G, Singh A. Alopecia areata: Pathophysiology, causes, and herbal treatment. Pharm Aspire 2021;13(4):144-150.

Source of Support: Nil,

Conflicts of Interest: None declared.

ABSTRACT

Alopecia areata is a pervasive immune system skin disease bringing about the deficiency of hair on the scalp and somewhere else on the body. It generally begins with at least one little, round, smooth patch on the scalp and can advance to add up to scalp going bald (alopecia totalis) or complete body balding (alopecia universalis). The scalp is the most ordinarily affected region; however, the facial hair or any hair-bearing site can be influenced alone or along with the scalp. Alopecia areata happens in males and females from infants to elders, however, beginning frequently happens among children. Over individuals, 147 million worldwide have, had, or will develop alopecia areata eventually in their lives. As indicated by Ayurveda, pitta dosha is the essential driver of balding. Pitta dosha could be turned around through diet and way of lifestyle. Herbal medication for balding contains herbs that can control hair fall and further develop hair growth.

Keywords: Alopecia areata, pathophysiology, drug-induced alopecia, herbal management

INTRODUCTION

Hair loss, commonly referred to as alopecia or baldness, is the loss of hair on one's head or body. At the very least, the head is usually engaged. Hair loss can range in intensity from a small patch to the entire body. Usually, there is no inflammation or scarring. Some persons experience psychological anguish as a result of hair loss. Alopecia areata, male- or female-pattern hair loss, and telogen effluvium, or hair thinning, are all common kinds. Male-pattern hair loss is caused by a mix of heredity and male hormones; female-pattern hair loss is unknown; alopecia areata is caused by an autoimmune reaction and telogen effluvium is caused by a physically or mentally stressful event. Following pregnancy, telogen effluvium is fairly common. Hair tugging, certain drugs such as chemotherapy, HIV/AIDS, hypothyroidism, and malnutrition, including iron deficiency, are all less prevalent causes of hair loss without inflammation or scarring. Fungal infection, lupus erythematosus, radiation therapy, and sarcoidosis are all causes of hair loss associated with scarring or inflammation. Hair loss is diagnosed in part based on the afflicted areas. Accepting the condition and shaving one's head may be all that is required to correct pattern hair loss. The drugs minoxidil (or finasteride) and hair transplant surgery are two options to consider. Steroid injections in the affected area can help with alopecia areata, but they must be repeated regularly to be effective. Hair loss is a very prevalent issue. Hair loss affects roughly half of males and a quarter of women by the age of 50. Alopecia areata affects about 2% of people at some point in their lives.[1]

Access this article online	
Website: www.isfcppharmaspire.com	**P-ISSN:** 2321-4732 **E-ISSN:** XXXX-XXXX

PHYSIOLOGY OF HAIR LOSS

Except for the palms and soles of the feet, human skin has a vast number of hair follicles. Hair follicles create two forms of hair after birth, depending on the location of the body: vellus hair and terminal hair. Vellus hair is soft and covers the whole-body surface that saves the palms and soles (usually <2 cm in length) (Headington 1984). The terminal hair is coarse and dense. ferocious It has a medulla and is normally colored. During birth, it is found in the scalp, brows, and mouth. A pair of lashes Every follicle has the ability to produce vellus hair first, followed by terminal hair.[1,2]

Each hair follicle passes through a three-stage cyclic activity cycle: anagen, catagen, and telogen (Rook and Dawber 1982). During the anagen phase, which can span anywhere from a few months to many years, the follicle continues to create hair. Anagen follicles are made up of three parts: the infundibulum, isthmus, and bulb are the three sections of the bulb, which include the dermal papilla and hair matrix. It 'is time to make the hair shaft. Normal hair follicles are in anagen the bulk of the time. The length of time that anagen lasts varies in different areas of the body and various persons. The length of many hair kinds is determined by the duration of anagen (scalp hair, axillary hair, and eyelashes). On an adult's scalp, the anagen phase lasts 4–8 years. High levels of mitotic activity define anagen follicles, rendering them particularly sensitive to harmful events. Catagen is a transitional stage. This lasts around 2 weeks. During catagen, hair follicles undergo retrograde changes, resulting in baldness. The resting period telogen is the resting phase of the hair cycle, which lasts about 100 days. An expanded base known as the club maintains the hair anchored inside the follicle during the telogen stage. Telogen hair appears only after the follicle re-enters anagen and produces a new hair shaft. Since their metabolic and mitotic activity is uneven, catagen and telogen follicles, unlike anagen follicles, are not responsive to noxious events. The ratio of anagen to telogen hair on a typical scalp is around 9:10. The amount of scalp hair lost daily varies between 30 and 100. Human hair development, on the other hand, is a slow process that shows seasonal variation, with hair loss peaking in late summer (Randall and Ebling 1991). Only when follicles are actively developing are they vulnerable to pathogens (Kligman, 1961). The hair matrix's mitotic activity is so high during the anagen phase that it may be likened to the body's most actively replacing tissues, such as bone marrow and mucous membranes. As a result, the anagen hair matrix is extremely vulnerable to noxious events, but the catagen and telogen hair matrixes are not. Since their mitotic activity is halted, follicles are largely harmless. As a result, the areas of the parts of the body with the most anagen follicles, such as the scalp and beard, are more severely affected. Exogenous insults, like medicines, have a greater impact on the body's lowest-lying parts eyebrows and eyelashes have a high proportion of anagen follicles. Hair follicles predictably respond to external assaults. The severity and duration of exposure to the noxious event, rather than the qualities of the agent(s) involved, determine the outcome (Reb ore). Hair follicles respond to foreign substance insults in a predictable way, but the outcome is more reliant on the severity and complexity of the circumstance. Rather than the qualities of the agent(s) involved (Reb ore), the duration of exposure to the noxious event is more important in the year 1993.[2,3]

Chronic telogen loss, on the other hand, may trigger or accelerate androgenic alopecia in genetically susceptible patients if the medicine that causes telogen effluvium cannot be stopped. In such circumstances, topical therapy is recommended. Minoxidil could be beneficial. Anagen effluvium is a common side effect of anticancer medications. Acute injury to rapidly dividing hair matrix cells is produced. Table 1 shows a list of medicines that can cause telogen effluvium. Hair loss is a common occurrence. This may be linked to anagen effluvium in some circumstances.[3]

DRUGS OR DRUG CLASSES THAT MAY INDUCE TELOGEN EFFLUVIUM

Allopurinol, androgens, bromocriptine, indomethacin, contraceptives (oral), nicotinic acid, potassium thiocyanate, propranolol, ibuprofen, retinol (Vitamin A) salicylates, sulfasalazine, cantharidin, cholestyramine, chloramphenicol, cidofovir, cimetidine, clonazepam, clotrimazole, colchicine, amiodarone amphetamines, buspirone, butyrophenones, danazol, diclofenac, dixyrazine, dyazoxide ethambutol, ethionamide, gentamicin, glatiramer acetate, glipalamide, gold salts, immunoglobulins, indandiones, indinavir, pentosane, polysulphate, phenindione, piroxicam, nitrofurantoin, octreotide, olanzapine, tacrolimus (oral), tamoxifen, terbinafine, terfenadine, and thiamphenicol.[4]

DRUG-INDUCED HAIR LOSS

Anticoagulants

Anticoagulants of any kind might cause hair loss. Heparin, heparinoids, coumarins, dextran, and indandiones are among them. Telogen effluvium affects around half of all patients, and it appears to be linked to treatment dosage. Women are more likely to experience it (Cornblatt and Hoit 1957).[3-5]

Antithyroid drugs

Lactogenic hypothyroidism, which occurs after thyrotoxicosis treatment, is frequently associated with reversible baldness. It can appear in persons who do not acquire iatrogenic hypothyroidism on rare occasions (Frenkel and Freinkei, 1972). Hair dryness is a common symptom of telogen effluvium. Brittleness and brittleness are two words that come to mind while thinking about this iodine, thiouracils, and other antithyroid medications may cause telogen effluvium. Carbimazole is a drug that is used to treat diabetes.

Oral contraceptives

After discontinuing oral contraceptive therapy, telogen hair loss is common for 2–3 months afterward. Increased telogen loss is solely a result of the higher percentage of anagen hair retained during pregnancy, as is the case with postpartum hair loss (Wong and Ellis). The year was 1984. Since estrogens cause the anagen phase to be prolonged, the use of contraceptives is recommended. This effect is only sometimes linked to the presence of low levels of estrogens.[5]

Lithium

Hair loss is a side effect of lithium carbonate therapy, and it can appear as early as a few months after starting treatment (Dawber and Mortimer, 1982). Telogen effluvium, which affects roughly 10% of patients, does not appear to be dose related (Orwin, 1983).

Antineoplastic agents (anti-cancer agents)

The most prevalent cutaneous adverse effect of antineoplastics is hair loss (anagen effluvium). Patients getting combination chemotherapy

Table 1: Different type of plants name, family, their local names, uses

Binomial name	Family	Local name	Mode of administration	Uses
Acacia sinuate (Lour) Mer.	Mimosaceae	Shigaki Soap pod	Leaves, bark of the plant are dried, ground into powder	Hair softer, strengthen hair root, lesser hair fall
Acalypha indica L.	Euphorbiaceous	Kuppameni	Whole plant extract is mixed with Coconut oil for use	Hair loss and baldness, lesser hair fall
Acorus calamus L.	Araneae	VA Sambu	The powdered root is mixed with coconut mild applied on the pat of the head[16]	Prevention of hair loss, lesser hair fall, strong hair follicles
Capsicum annum L.	Solanaceae	Melagi	Fruit paste is applied to the hair to the affected area of the head	Stimulates hair growth, strong hair follicles
Cocos nucifera L.	Arecaceae	Thennai	Coconut oil with Curry leaves, coconut milk	Black hair, strengthens hair, prevents breakage
Ficus racemosa L.	Moraceae	Arasa maram	Seed powder with Coconut oil applied to the white hair	White hair makes black
Hibiscus rosa sinensis L.	Malvaceae	Semparuthi	Flower extraction is Boiled with Coconut oil	Stops hair fall and graying of hairs
Piper betel L.	Piperaceae	Vettrilai	Grinding leaves with sesame oil are applied to the scalp	Control hair loss, Strengthen hair follicles
Withania somnifera (L.) Dunal.	Solanaceae	Ammukkara	Powdered leaves are boiled with water and applied	Control hair loss, It turns gray hair into white
Wedelia chinensis (Osbeck)	Asteraceae	Bhringraj	Leaves are crushed and applied on the roots of the hairs	Promote hair growth, strengthen hair
Ocimum sanctum L.	Labiatae	Tulsi	Powdered leaves are boiled and applied on the roots of hairs	Prevent hair loss, avoid dandruff , premature graying
Glycyrrhiza glabra L.	Fabaceae	Mulethi	Roots are removed from plants and applied on the hairs roots	Help fight dandruff, stimulate hair growth[17]
Nerium oleander L.	Apocynaceae	Kaner	Bark, roots both are applied on the roots of the hairs by massaging	Stimulate hair growth, Anti hair fall, anti dandruff
Terminalia chebula Retz.	Combretaceae	Harad	Fruits juice and extract can be applied on hairs	Control hair loss and promotr hair growth
Rubia Cardi folia L.	Rubiaceae	Manjistha (Indian Madder)	Fruits, leaves both can be applied on the roots of hairs	Manage hair problem like graying of hair
Jasminum officinale L.	Oleaceae	Mallika	Leaves can be applied in juice form on hairs	Anti-lice agent, keep the scalp moisturized
Calacanthus grandiflorus Mill	Acanthaceae	Sahchar	Leaves can be applied on the hairs	Promotes hair growth[17]
Trichosanthus species	Cucurbitaceae	Patol	Leaves, fruits both can be applied in juice form	Manage hair problem of gray hair
Ribes rubrum L	Grossulariaceae	Dakh	Flowers, Leaves both can be applied on hairs	Say no to hair loss with black raisins
Pterocarpus marsupium Roxb.	Leguminosae	Asana	Heartwood Stem bark applied on the hairs	Promote blood circulation, hair growth
Eclipta alba Hassak	Asteraceae	Bharngaraja	Whole plant can be applied in juice form on the hairs	Treats dandruff , treat baldness
Mussa paradisiacal Linn.	Musaceae	Kadali	Rhizome can be applied in juice form on the hairs	Manage hair, control dandruff
Cardiospermom halicacabom Linn	Sapindaceae	Karnasphota	Seeds, Roots can be applied in juice form	Treat dandruff and alopecia
Ficus religiosa Linn.	Moraceae	Pipal tree Ashwati tree	Bark, Ethanolic extract can be applied in juice form	Antiseptic and healing properties, promote hair growth
Nordostachys jatamansi DC	Caprifoliaceae	Spike nard	Rhizome, Roots, Volatile oil, Ethanolic extract	Smooth silky and healthy hair, control dandruff.[17,18]

experience more frequent and severe hair loss than those getting a single medication. The severity of hair loss differs across people on the same treatment plan. Anagen hair loss occurs in the majority of patients after the first or second cycle of chemotherapy administration, or 1–8 weeks following the commencement of treatment. Telogen hair loss is a common symptom.[5]

Antiretroviral medications

In up to 10% of patients, severe telogen effluvium and patchy hair loss resembling alopecia areata are common side effects of indinavir therapy. Due to the elevated plasma concentration of the medicines, combined treatment has been linked to severe hair loss. Legs, tights, pubic, thoracic, and axillary hair are frequently involved with this condition.[6]

Interferons

Interferons are proteins that play a role in the immune system. Interferon-induced telogen effluvium affects up to 50% of individuals and is not dose related. Hair shedding is reversible following treatment interruption and, in certain situations, even if treatment is continued. IFN injection sites can also cause transient localized alopecia.[6,7]

Minoxidil

Telogen effluvium develops 2–3 months after topical minoxidil is stopped. Hair loss is common and is caused by the simultaneous telogen entrance of all follicles that had grown longer under the influence of minoxidil. Hair loss can occur at any time during minoxidil medication; this could be related to the fact that the onset of anagen triggered by minoxidil causes the old club hair to detach.[8]

Psychotropics

Up to 20% of long-term lithium users have hair loss. This could potentially be a result of hypothyroidism caused by lithium. Lithium consumption has also been linked to hair straightening. Hair loss is a common side effect of valproic acid and divalproex, which can affect people receiving large doses. Telogen effluvium is a typical side effect of fluoxetine and paroxetine use.[7,8]

Radiation

Alopecia areata-like temporary patchy alopecia is a possible side effect of neurosurgery procedures involving fluoroscopic imaging. The retroarticular areas are the most typically affected, as they get the largest doses (300–600 Gy).

Retinol (Vitamin A)

Hair loss is caused by high doses of Vitamin A. Mild hair loss is common in individuals who use Vitamin A supplements, according to our experience. Vitamin E supplementation has been shown to promote Vitamin A toxicity.[8]

Retinoids

In up to 20% of people, acitretin, etretinate, and isotretinoin induce hair loss with obvious alopecia. The negative effect is dose-dependent and can damage body hairs as well. Alopecia can be very severe in some cases, and total alopecia has also been reported. Early Telo ptosis is likely to have a role in retinoid-induced hair loss.[8]

Drugs inducing hair graying

Atheism, benzoyl peroxide, butyrophenones, chloroquine, cyclosporine A, etretinate, hydroquinone, interferon, methenamine, phenols, phenylthiourea, and triperidol.

PHYLOGENY AN ETIOLOGY AND PATHOPHYSIOLOGY OF HAIR LOSS

Baldness is a natural trait associated with sexual maturity in many primate species, including humans. The stump-tailed macaque has been the most extensively used animal model since the process is visible in both sexes. The research on this model was the first in-depth analysis demonstrating the progression of terminal follicles to secondary vellus units.[9,10]

PREVALENCE AND GENETICS

Although the prevalence of androgenetic alopecia in any population has not been adequately studied, it likely approaches 100% among Caucasoid races. This means that some degree of change of terminal to vellus follicles on the vertex from adolescence onward is a universal occurrence. Negroids are less likely to go bald than Caucasoids, with Mongoloid races being the exception. The least harmed before Hamilton's research, investigators could only make qualitative judgments of hair. He looked at 312 white males and 214 white females between the ages of 20 and 89 and offered a grading system. The following are examples of baldness that are nonetheless useful.

- Type 1: A full head of hair
- Type 2: Bitemporal recession is a type of recession that occurs across a period
- Type 3: Borderline
- Type 4: Deep frontotemporal recession with some midfrontal recession is common. In the past, this level of frontotemporal loss may be linked to some vertical problems in some people
- Type 5: Frontotemporal recession is increased, and the vertex is markedly denuded
- Type 6: Loss has increased in both areas, which are merging. Only a ring of scant hair separates
- Type 7: Enlarged frontotemporal and vertical portions
- Type 8: Complete confluence of both zones.

Before puberty, Type 1 was a normal scalp in both sexes, but after puberty, Type 2 took over in 96% of men and 79% of women. 58% of men aged 50 and up had Type 5–8 scalps, and the extent of baldness tended to rise until the age of 70. By the age of 50, around a quarter of women had developed Type 4 scalps. There was no additional growth, and no women with Type 5–8 were detected.[11,12]

PATHOLOGY

Following a period of increased shedding, typical scalp hair follicles miniaturize, resulting in a decrease in hair cycle time and hence more telogen transformation. Hypoplasia is seen histologically, not follicular atrophy or "drop off." Many people have seen inflammatory changes. The writers are most likely suffering from seborrheic dermatitis. There were no specific histopathological abnormalities found despite its physiological role in follicular regulation, have been found in the dermal papillae expansion.[13]

PATHOGENESIS

Although our understanding of the pathophysiology of common baldness is far from comprehensive, trustworthy data are constantly increasing. A genetic predisposition is required, as well as androgen production. In ten eunuchoid, castrated guys, Hamiliton's famous study revealed that no baldness developed. Before puberty, and in 34 males who had had their orchids removed throughout their youth; the prevalence of obvious baldness among these 54 adult men was 40%. When testosterone was given to the patient, when testosterone levels were low, frequent baldness appeared in genetically susceptible people. When the medication was stopped, the baldness did not progress, but it also did not go away. Since then, these findings have been confirmed. Many researchers have looked at the decrease and transit of androgens from the adrenal glands (both sexes), ovaries, and testes. The failure of investigators to standardize the schedule of sampling, with normal controls being included when in doubt, impeded most of the early investigations. In truth, they differ only in degree from affected persons; this is especially true in women. Generally speaking, one can conclude that the key genetic component is the level of circulating androgens, whether free or total. Organ response: in this case, follicular receptivity and androgen reactivity, which must be present at the matrix/papilla unit's end. The enzyme 5-alpha-reductase in receptor tissue has been widely investigated in its conversion of testosterone to the powerful androgen dihydrotestosterone (DHT).[13,14]

Free testosterone, sex hormone-binding globulin, and dehydroepiandrosterone have all been studied as biochemical and endocrinological anomalies that may be linked to androgenetic alopecia. Conclusions about their relevance cannot be reached just now, and they will have to wait. Larger, more precisely controlled clinical trials are awaited. These were tested in acne-free, non-hirsute women. Since the significance of modest alterations in androgenetic alopecia is unknown, it is generally not advisable. However, prolactin is required for women with androgenetic alopecia, amenorrhea, and/or galactorrhea. To rule out the presence of a pituitary adenoma, a measurement is taken.

Hair loss, commonly referred to as alopecia or baldness, is the loss of hair from a specific area of the head or body. At the very least, the brain is involved. Hair loss can range in intensity from a small patch to the entire body.

Gray or white hair is the outcome of changes in the hair that cause it to turn white. As we become older, we gradually change our hair color. It normally starts with the temples, then moves to the back of the head, and finally all of the hair. This procedure affects 65% of women and is more common in women than in men. It generally affects women over the age of forty, while only half of the men are affected. The plants that are used are all natural. In the accompanying gatherings, hair-care specifics might be arranged:

(1) Plants used as hair chemical, (2) plants used as hair coloring operator (sick), (3) plants used as anti-dandruff, and (4) plants used as hair tonic/hair health development.[15]

MATERIALS AND METHODS

The details of collected plants with their scientific name, family, local name, mode of administration, and uses are presented in Table 1.

ALOPECIA AREATA (NON-SCARRING ALOPECIA)

Definition

Non-scarring alopecia, also known as non-cicatricial alopecia, is hair loss that occurs without any scarring. Inflammation and discomfort are usually minor, but hair loss is severe. Scarring hair loss, on the other hand, occurs when hair follicles are replaced with scar tissue as a result of inflammation. Hair loss can occur all over the scalp (diffuse) or in specific areas (focal). The loss may be abrupt or gradual, and it may be accompanied by stress. Due to the effects of DHT on the hair follicles, androgenetic alopecia, also known as male pattern or female pattern hair loss, is the most common cause. Hormonal impacts, age, diet, autoimmunity, mental stress, physical stress, pharmacological effects, heredity, or infections are all possible causes of this illness.[19,20]

Non-inflammatory forms of alopecia

Type	Significant features	Treatment and comments
Anagen effluvium	Diffuse hair loss days 2 weeks after exposure to a chemotherapeutic agent; incidence after chemotherapy is estimated at 65%	No pharmacologic intervention has been proven effective; scalp cooling is not recommended
Telogen effluvium	Clinical characteristics are used to diagnose telogen effluvium. Hair thinning affects the entire scalp as well as the hair on other parts of the body. Diffuse thinning with short hairs of normal thickness and no localized areas of complete alopecia[21]	Minoxidil is an over-the-counter medication that may be used to treat hair loss Avoid over-vigorous combing, brushing, and any form of scalp massage by gently manipulating the hair[20]
Trichotillomania	An overpowering desire to pull one's hair out continuously. The scalp hair is the most commonly affected. Eyelashes, brows, and even the beard might be affected.[21]	Cognitive-behavioral therapy (CBT) Acceptance and commitment therapy (ACT).[22]
Traction alopecia	Hair that has been tightly braided or pulled into ponytails or cornrows for an extended period. Breakage and gradual hair loss are caused by chronic strain on the hair shafts[23]	Antibiotics to keep any open sores from becoming infected Topical steroids are used to reduce edema on the scalp Shampoos with antifungal properties[21,23]
Temporal triangular alopecia	It appears as an alopecic patch that is usually limited to one frontotemporal region of the scalp[24]	If the lesion is tiny, surgical removal or hair transplants are options.

Inflammatory forms of alopecia

Type	Significant features	Treatment and comments
Psoriatic alopecia	Follicular hyperkeratosis, a rise in the number of telogen hairs, and a perifollicular lymph histiocytic cell infiltrate surrounding	Topical steroids, vitamin D analogs, UVB narrow band, and methotrexate were used to treat the condition[25]
Alopecia areta	Short, vellus hairs, yellow or black spots, and broken hair shafts are found on inspection of acute, patchy hair loss[26]	Intralesional triamcinolone acetonide injection intradermally
Androgenetic alopecia	Hair loss runs in the family; the condition worsens over time Men: frontal and vertex scalp thinning total hair loss Women: widespread vertex hair thinning with a frontal hairline sparing[27]	Men topical minoxidil (2% or 5% solutions) women topical minoxidil (2% solution)
Syphilitic alopecia	A rare complication of secondary syphilis SA can appear in a diffuse form that looks like telogen effluvium or a moth-eaten form that looks like a variety of diseases	For patients without immunocompromise, a single intramuscular injection of 2.4 million units of benzathine penicillin G is recommended; nevertheless[29,30]

PATHOLOGY OF REGROWING WHITE HAIRS

Alopecia areata has been linked to changes in the dermal papilla. In alopecia areata, Van Scott and Ekel (1958) observed an increase in the ratio of papillary to matrix cells in anagen follicles. Some of the follicles we examined also revealed this alteration, which we believe was caused in part by the presence of estrogen. Inflammatory cells are a type of cell that causes inflammation. The majority of bulbs, however, have cellularity in the papilla and matrix. Even when tissue damage was apparent, it appeared normal. As a result, we propose that alopecia areata is a disease of the cortical keratocyte that is differentiating. We can explain a variety of clinical and pathological characteristics of alopecia areata using this idea. An attack on the government. In the anagen follicle, differentiated cortical keratocytes cause a localized interruption of keratinization. When the hair shaft reaches the skin's surface, it fractures. The follicle is unable to continue in its current state. It exits anagen and enters catagen.[31]

The process of de-differentiation from catagen to telogen proceeds normally, and a club is generated (Fckert, Church and Ebling, 1968). The fact that hair shafts are still being produced during this stage of involution implies that matrix activity is not abruptly ceasing stopped. Despite this, cortical cells still have the ability to produce keratin. The follicle is in telogen when it is inactive, probably immune to attack, which could explain the disease's non-destructive character. Anagen is a term used to describe a matrix activity that resumes, but anagen formation is halted at the same level as before III/IV anagen (Van Scott, 1958). Cortical cells begin to differentiate at this stage of anagen (Chase, Rauch and Smith, 1951). The pigmentary disruption in regrowing white hair is best explained as early anagen melanocyte activation that is partial or incomplete. This could just be a toxic response to local inflammation, even though there was no link between impairment of the immune system and impairment of the immune system. pigmentation and the amount of inflammatory cells invading On the other hand, an attack on the target could be directed towards the target. Cortical keratocytes' ability to receive pigment and maybe start reactions may be harmed as a result. the activity of melanocytes A further option is that the attack is targeted precisely at systemsThe cortical keratocyte is a type of keratocyte that regulates pigmentation. The epidermal keratocyte is thought to have an important role in the modulation of pigmentation.[33]The symbiotic interaction between cortical keratocyte and hair bulb melanocyte is particularly noticeable in the hair follicle. The telogen bulb contains a limited number of "dormant" melanocytes (Sugiyama and Kukita, 1976). Anagen III causes functional activation of melanocytes, and the pigment is transported to the Cortex keratocytes. During anagen (kukita,1957), intense pigmentary activity continues till the approach of the end of the catagen. Pigmentation loss occurs before other signs of catagen involution, such as the appearance of wrinkles telogen club hair's proximal white tip (Montag and Parakkal, 1974). As a result, melanocyte activity closely resembles and may even be dependent on cortical differentiation. The presence of functioning melanocytes in the top half of the anagen bulb and their near absence in the lower bulb is due to structural differences. Cortical differentiation is also required for pigment transfer, according to the matrix. It 'is possible that senile graying develops due to a decrease in the number of melanocytes rather than wear and tear on the melanocytes.[34]The ability of cortical cells to activate melanocytes. If the disease attack in alopecia areata is indeed alopecia areata, it is aimed on the keratocyte's pigmentary mechanism, which would explain the senile white hairs are sparse. Indirect evidence for this concept comes from a recent vitiligo study in which degenerative changes in the basal keratocytes in non-lesional skin were observed, identical to those reported in cortical cells in alopecia areata (Moellman *et al.*, 1982). This, according to the authors, is due to melano cytotoxicity which is a term used to describe the toxicity of me the melanocytes, on the other hand, looked to be unharmed. In the end, because of the variety of clinical and pathological manifestations of alopecia areata, it has been claimed that it is not a single clinicopathological entity. Clinical patterns and illness correlations vary widely (Ikeda, 1965). The sparing of senile white hair is a rare occurrence. In addition, there is a variable feature. The idea that alopecia areata is a disease of differentiating cortical keratocytes encompasses this idea because the hair follicle would have a similar reaction pattern regardless of the disease's precise origin.[36]

Hair deficiencies and supplements

Deficiency	Food sources	Treatment	Mode of administration
Protein	Eggs, berries, shrimp, fatty fish, tofu, legumes, nuts, and cheese[38]	As it has both antibacterial and antifungal properties to treat the disease	Leave hair products, in the form of a mask, shampoo, conditioner[37]
Zinc	Oysters, beef, spinach, wheat germ, pumpkin seeds, and lentils	Zinc parathion has antibacterial, antimicrobial and can inhibit the yeast	zinc supplementation in form of tablets and capsules and also some syrups
Vitamin B12	Milk and dairy Products, eggs, nuts cheese Fish[39]	Keratin treatment, cut the damaged hair so that growth can be increased	Shampoo and conditioner and some other products apply on scalp or hairs
Biotin	Egg yolks, peas, beans, and lentils are high in protein, fiber	oral marine protein supplement (MPS) in the form of tablets are available	Supplements, shampoo, conditioner[39]
Vitamin C	Blackcurrants, blueberries, broccoli, guava, kiwi fruits, oranges	Removal of Malassezia fungus on the scalp, especially on the roots of hairs	Supplements, tablets, different oils for hair scalp
Niacin	Lean meats; poultry; eggs; seafood; beans, peas, and lentils; nuts and seeds	Medications and natural remedies which Improve Blood circulation to the scalp	Tablets, oils, shampoo, and some other forms are available in the market
Selenium	Brazil nuts, seafood, and organ meats	Homeopathic medicine or supplements	Supplements, conditioner, serum
Copper	Shellfish, seeds and nuts, organ meats, wheat-bran cereals, whole-grain	Copper sulfate, copper peptides in the form of the liquid dosage form	Serum, shampoo, conditioner and some other forms are available
Vitamin A	Cheese. Eggs. Oily fish, fortified low-fat spreads.[41]	Sebum moisturizes the scalp, supplements, natural products	Oils, the serum is available in the market at affordable prices to people[40]
Vitamin D	Fortified soy milk. Mushrooms, Fortified cereals. Fortified orange juice	Supplements doctors best vegan d3, country life vegan d3	Oral intake of supplements, medicines, serums

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