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The Etiological Correlation of Seven Common Types of Alopecia

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Abstract:

In recent years, the population suffering from alopecia in China has been increasing, which severely affects the daily life and mental health of individuals, making it one of the diseases that cannot be ignored. The occurrence of alopecia is associated with various factors, including genetic predisposition, induction by other diseases or medications, and environmental influences. Current research on the specific etiology of alopecia is not comprehensive, which hinders the diagnosis and treatment of various types of alopecia and the inhibition of hair loss from its roots or progression. This article summarizes several types of alopecia with high incidence rates, such as androgenetic alopecia, alopecia areata, frontal fibrosing alopecia, and seborrheic dermatitis, aiming to elucidate the specific causes of different types of alopecia and to organize the relationships between various forms of alopecia. This work lays a solid foundation for the diagnosis and treatment of alopecia by considering potential common etiologies and proposing three biological indicators for triggering alopecia: hormones, microorganisms, and the "common pathway" – immune dysregulation.

Keywords: Alopecia, Etiology, Follicular Immunity.

1. Background:

Alopecia is a degenerative hair follicle disorder caused by various factors, characterized by localized or generalized hair loss. It can be classified into scarring and non-scarring types and is associated with genetic factors, lifestyle habits, psychological stress levels, medication use, environmental pollution, gender, and other factors, exhibiting significant individual variability. As human civilization progresses into the modern era, alopecia has become increasingly prevalent among younger populations and affects a broader demographic [1]. Meanwhile, treatment technologies for alopecia are rapidly advancing, with progress in exosome therapy [2], hair transplantation [3], and novel drugs targeting new pathways [4, 5]. However, for the development of treatment methods, etiological research is crucial. This article aims to analyze the causes of seven common types of alopecia (androgenetic alopecia, frontal fibrosing alopecia, telogen effluvium, lichen planopilaris, central centrifugal cicatricial alopecia, alopecia areata, and folliculitis decalvans) to explore similarities in their pathogenic pathways and identify potential common targets. This will provide new insights for developing innovative therapies and enhancing the universality of existing treatments.

2. Etiological Analysis of Seven Common Types of Alopecia

2.1 Androgenetic Alopecia (AGA)

Androgenetic alopecia (AGA) is a non-scarring form of hair loss characterized by progressive reduction of hair follicles or the appearance of non-functional or dead follicles in a specific pattern on the scalp. Hormonal factors, genetics, micronutrient deficiencies, microinflammation, and stress are all implicated in its development [6]. AGA is the most common type of alopecia, accounting for approximately 90% of all

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hair loss cases. Typical symptoms include thinning hair starting from the frontal and temporal regions, receding hairline, and gradual extension to the vertex. This condition often has a familial predisposition, with significant gender differences—21.3% in males and 6% in females. AGA also exhibits racial disparities, with higher prevalence among Caucasians than among Asians and Africans. The incidence of AGA is age-dependent, beginning at puberty and increasing with age. It is non-contagious and often accompanied by excessive sebum production [7]. The causes of AGA include the following:

2.1.1 Genetic Factors

Susceptibility to AGA is primarily determined by genetics and is considered a polygenic disorder. Androgens are the most critical regulators of human hair growth, with varying effects in different body regions due to differences in gene expression responses to androgens. In the scalp follicles of susceptible males, androgens inhibit hair growth [8]. The *StuI* polymorphism is linearly correlated with AR activity and is associated with AGA. Variants in the *EDA2R* gene also increase susceptibility to AGA [9]. Studies have identified genetic susceptibility loci for AGA on chromosome 7p21.1, 3q26 (*HDAC9*), 20p11 (*PAX1/FOXA2*), and the X chromosome (androgen receptor/*EDAR2*).

2.1.2 Androgen Receptor (AR) and 5α-Reductase Factors

Research indicates that circulating androgen levels in AGA patients are normal, but increased AR expression makes scalp follicles highly sensitive to these hormones. 5α-reductase has two isoforms (type I and II), which catalyze the conversion of testosterone (T) to dihydrotestosterone (DHT). DHT has a fivefold higher affinity for AR than T, and both isoforms play significant roles in androgen metabolism [10].

2.1.3 Cellular Senescence Factors

Hair follicle stem cells (HFSCs) and dermal papilla cells (DPCs) play dominant roles in follicular morphogenesis and cyclic hair growth [11, 12]. DHT, the most potent androgen in hair growth, acts on DPCs, which mediate signaling by secreting growth factors and extracellular matrix components [13, 14]. DHT induces DPCs, leading to progressive follicular miniaturization, shortening the anagen phase and prolonging the telogen phase, ultimately resulting in baldness. Senescent HFSCs exhibit impaired ability to enter the hair growth phase, Wnt-inhibited hair growth signaling, and DNA damage-triggered senescence, leading to follicular miniaturization. Changes in HFSC subpopulations have also been observed in AGA [15].

2.1.4 Other Disease Factors

Risk factors for coronary artery disease (CAD), such as low HDL, high LDL, very low-density lipoprotein, triglycerides, serum lipoprotein-a, and serum homocysteine, increase with the severity of AGA. The risk of CAD in AGA patients rises with the grade of AGA [16]. Conditions like benign prostatic hyperplasia, prostate cancer, and CAD are more common in AGA patients than in non-bald individuals [17].

2.1.5 Emotional Factors

The onset of AGA also depends on psychological and emotional factors. Compared to the general population, psychiatric disorders are more prevalent among individuals with hair loss.

2.2 Frontal Fibrosing Alopecia (FFA)

Frontal fibrosing alopecia (FFA) is a band-like scarring alopecia in the frontal-temporal scalp, classified as a special form of lichen planopilaris. It primarily affects postmenopausal women, though its incidence in men may be underestimated due to potential overlap with androgenetic alopecia [18]. Dermoscopy of the eyebrows reveals "road sign" patterns, hairs growing in different directions, and the presence of black, yellow, and gray dots, diffuse erythema, and follicular unit loss [19]. Histological examination shows abundant perifollicular and infundibular lymphocytic infiltration, hyperkeratosis of the infundibulum and follicular ostia, and apoptosis and vacuolar degeneration of basal keratinocytes [18]. The etiology remains unclear, with potential links to sex hormones, genetic predisposition, autoimmunity, environmental factors, defects in lipid metabolism, and neurogenic inflammatory responses [20]. Hormonal changes during pregnancy, breastfeeding, hysterectomy, or hormone/raloxifene therapy, as well as exposure to sunscreen ingredients, moisturizers, or sunlight, may also play a role [18].

2.2.1 Pathogenesis

Immune cells and cytokine-mediated inflammatory responses appear to play a significant role in FFA [21]. Increased infiltration of CD8+ cytotoxic T cells and dendritic cells around damaged follicles (particularly near the bulge region) exacerbates inflammation, leading to the collapse of immune privilege (IP) and damage to epithelial hair follicle stem cells (eHFSCs) [22]. This results in progressive fibrosis of the entire follicular unit, causing scarring alopecia [23].

2.2.2 Genetic Factors

Research has focused on the role of human leukocyte antigen (HLA) in FFA development. The HLA-B*07:02 mutation appears to promote an autoinflammatory response against eHFSCs, significantly increasing the risk of FFA [24]. A study of FFA patients identified that 83.8% carried the rs9258883 polymorphism in HLA-B*07:02, with most lacking the protective rs1800440 polymorphism in *CYP1B1* (75.2%) [25]. Alterations in genetic pathways such as PPAR-γ and mTOR are also associated with FFA. These pathways influence lipid metabolism, sebocyte differentiation, and immune responses, potentially affecting follicular

health through inflammation and fibrosis induction. Reduced PPAR-γ activity leads to fibrosis and inflammatory cell infiltration, while mTOR signaling interacts with PPAR-γ to regulate lipid homeostasis and inflammatory processes [23]. Specifically, decreased peroxisome and cholesterol production may cause the accumulation of pro-inflammatory lipids in hair follicles, leading to inflammatory cell infiltration in the bulge region [26].

2.2.3 Environmental Factors

Exposure to light, particularly ultraviolet radiation, has been hypothesized to contribute to FFA [27]. Light may influence the synthesis of certain compounds, such as 6-formylindolo[3,2-b]carbazole (FICZ), which can have pro- or anti-inflammatory effects depending on their concentration. Retrospective studies using surveys and clinical cases have also suggested a link between FFA and the use of sunscreen and other facial cosmetics in both male and female patients [28, 29].

2.2.4 Hormonal Factors

Changes in hormone levels, particularly during menopause, may influence the onset and progression of FFA [23]. Reduced levels of DHEA and androgens can induce fibrosis in FFA [21]. Based on its frequent comorbidity with androgenetic alopecia and clinical improvement with anti-androgen therapy, some propose a hormone-dependent etiology for FFA [30].

2.3 Telogen Effluvium (TE)

Telogen effluvium (TE) is a scalp disorder characterized by diffuse, non-scarring hair loss [31], which is a reactive process triggered by metabolic stress, hormonal changes, or medications. Studies indicate that TE has no genetic cause [32].

2.3.1 Pathogenesis

TE is triggered when physiological stress causes a large number of hairs in the anagen phase of the hair cycle to abruptly enter the telogen phase. During this stage, telogen hair growth ceases for 1 to 6 months (average 3 months). When affected hairs re-enter the anagen phase, the telogen hairs are extruded from the follicles, resulting in noticeable hair loss. At the molecular level, etiological factors may disrupt the delicate balance of growth factors, neuroendocrine signals, and cytokines involved in follicular homeostasis. Such disturbances can lead to premature induction or prolongation of the catagen phase, accelerating the transition of hairs into the telogen phase. Relevant studies suggest that inflammatory mediators, oxidative stress, and changes in the follicular niche microenvironment contribute to the persistence of TE.

2.3.2 Inflammatory Response Factors

Surveys indicate that the proportion of TE patients among all hair loss types increased after the COVID-19 pandemic. In addition to direct damage to hair follicles by various viruses, pro-inflammatory cytokines can also harm follicles, and the coagulation cascade accompanied by microthrombi (blocking follicular blood supply) may form [33].

Viral infections trigger the intense release of pro-inflammatory cytokines. Viruses induce a robust antiviral response, particularly through interferons, which are TE-inducing molecules [34]. High levels of IL-6 act on hair follicles, leading to the collapse of immune privilege and inducing the catagen phase, causing localized inflammation. Other molecules elevated in COVID-19 include matrix metalloproteinases 1 and 3 and IL-1b, which may inhibit hair follicle growth [35]. Simultaneously, during high fever, cytokines initiate apoptosis of follicular keratinocytes, pushing them into the catagen phase and subsequently the telogen phase. Additionally, malnutrition, severe illness, and chronic wasting diseases can disrupt the hair growth cycle, causing premature entry into the telogen phase and immediate anagen release [36].

2.4 Lichen Planopilaris (LPP)

Lichen planopilaris (LPP) is a scarring alopecia that predominantly affects middle-aged women, with an estimated incidence of 1% to 7%. Patients present with hair thinning, which may be accompanied by scalp itching or tenderness [37]. It is characterized by a chronic and destructive inflammatory process [38]. LPP is now classified as a primary lymphocytic disorder based on lymphocyte, neutrophil, or mixed infiltrates [39] and is often irreversible [40].

2.4.1 Immune-Inflammatory Response Factors

Cell-mediated immunity can drive the clinical manifestations of LPP. The immune response primarily involves the bulge region, a continuous part of the outer root sheath rich in stem cells. The participation of T lymphocytes (CD4 and CD8) is activated by an increase in Langerhans cells in the dermis and epidermis. Reports indicate that Th17 cells (a subset of CD4+ T helper cells) also play a crucial role in promoting immune-inflammatory responses in autoimmune diseases [41].

2.4.2 Microbial Population Factors

One study demonstrated an imbalance in the scalp microbiota of LPP patients, which plays a role in its pathophysiology. Another study found that compared to healthy controls, the LPP group had increased abundance of Cyanobacteria and Euryarchaeota phyla, fewer Firmicutes, and higher microbial diversity [38].

2.4.3 Environmental Factors

Due to global climate change, particularly air pollution, certain members of the Cyanobacteria phylum have proliferated in the atmosphere. This is associated with higher cyanobacterial richness in LPP patients, and metabolites produced by this phylum have negative effects on human health [42]. Pollutants such as particulate matter and heavy metals may

accumulate on hair [43], inducing oxidative stress by increasing reactive oxygen species (ROS) production [44] and contributing to clinical conditions related to hair loss, including LPP [45].

2.5 Central Centrifugal Cicatricial Alopecia (CCCA)

Central centrifugal cicatricial alopecia (CCCA) is a scarring alopecia characterized by permanent patches of hair loss that begin at the vertex or crown of the scalp and gradually spread outward in a centrifugal pattern [46]. CCCA shows significant racial and gender predispositions: it most commonly affects individuals with tightly coiled or kinked hair, and 2.7% of African American women are affected by CCCA [47].

2.5.1 Genetic Factors

A positive correlation has been found between mutations in type III peptidylarginine deiminase and CCCA. This enzyme specifically acts on the deamination of proteins within the hair shaft. Loss of this function increases hair fragility, leading to hair loss [48].

2.5.2 Inflammatory Response Factors

Various inflammatory cytokines extracted from the scalps of women with CCCA indicate it is an inflammatory disease. Multiple studies have confirmed a positive correlation between a history of fungal infections and the development of CCCA. One study showed that STAT3 is activated in perifollicular lymphocytes of CCCA patients. STAT3 activation is significant for increasing Th17 cells, which secrete pro-inflammatory cytokines that may play a role in the progression of this disease [49].

2.5.3 Other Disease Factors

The incidence of CCCA is higher in women with a history of depression and anxiety [50]. Additionally, CCCA is associated with type 2 diabetes and uterine leiomyomas. Some studies found that CCCA patients have at least a fivefold increased risk of developing leiomyomas, particularly among women of American descent [48].

2.6 Etiology of Alopecia Areata (AA)

Alopecia areata (AA) is a common chronic tissue-specific autoimmune disease characterized by non-scarring hair loss with preserved hair follicles. The clinical presentation ranges from small patchy hair loss to diffuse or complete alopecia, potentially affecting the entire body surface. Biopsies reveal lymphocytic infiltration in the hair bulb or lower half of hair follicles during the anagen phase. Approximately 2% of the global population experiences AA at some point in their lives [51]. Depending on severity, it may lead to psychiatric disorders like depression and anxiety, as well as psychosocial issues [52]. Surveys indicate that while the global disease burden of AA improved between 1990-2019, it remains relatively high overall [53]. AA tends to be milder in elderly patients but more severe in children, often progressing to alopecia totalis/universalis [54]. The exact pathogenesis remains unclear, though current research provides evidence implicating genetic, immunological, and psychological factors.

2.6.1 Genetic Factors

Recent genome-wide association studies have linked AA to Treg cells, CTLA-4, IL-2/IL-21, CD25 (IL-2RA), ULBP6 (UL16-binding proteins), and NK cell receptor NKG2D. Polymorphisms in AIRE-207 (autoimmune regulator-207) and TNF/LTA (lymphotoxin-alpha) genes are also frequently observed in AA patients [54]. Genetic epidemiological studies show that 8.4%-25.0% of AA patients have a positive family history, with first-degree relatives having higher susceptibility. Compared to the general population, if either parent is affected, the first twin is more likely to develop AA regardless of other family members' status [55].

2.6.2 Immune Dysfunction

AA is recognized as a common type of immune-mediated hair loss where autoimmune attack on hair follicles causes non-scarring alopecia. The pathogenesis is attributed to collapse of hair follicle immune privilege (HF-IP) [56]. As an immune-privileged (IP) site, HF-IP breakdown is considered prerequisite for AA development, while its restoration may enable spontaneous or long-term remission. Therefore, accelerating HF-IP reconstruction represents a promising therapeutic approach. Local immunosuppressive molecules like TGF-β1, IL-10, α-MSH, IDO, and VIP may help maintain this immunoinhibitory microenvironment [57].

Additionally, human leukocyte antigens (HLA) significantly influence autoimmune diseases. AA patients exhibit markedly increased expression of HLA-A, HLA-B, and HLA-C - rarely seen in healthy individuals. Clinical observations also show AA patients frequently present with other autoimmune diseases [58], further supporting AA's autoimmune nature.

2.6.3 Mental Health Factors

Multiple studies demonstrate close relationships between AA onset and psychological stress/mental health status. While anxiety/depression may not play primary roles in AA pathogenesis among limited patient cohorts [56], exacerbated life stress can trigger disease onset/worsening. Complex interactions among insecure attachment, alexithymia, and poor social functioning may increase AA risk. The observed high alexithymia traits and avoidant attachment in AA patients reflect emotional regulation deficits. Though mental health is important, AA occurrence in infants/-neonates necessitates consideration of alternative factors.

2.6.4 Smoking Factor

As a classic inflammatory dermatosis, AA associates

with environmental stimuli including smoking. While the precise smoking-related mechanism remains unclear, cigarette smoke: Elevates proinflammatory cytokines (IL-17, IFN-γ) while reducing anti-inflammatory cytokines ,Activates Th17-mediated skin inflammation, Increases Th2 cytokine IL-13, exacerbating Th2-dominant immune responses [56,58] Thus, AA patients should strictly avoid smoking.

2.7 Folliculitis Decalvans (FD)

Folliculitis decalvans (FD) is a chronic, recurrent pustular folliculitis of the scalp, typically involving the central scalp with crusted plaques and centrifugal progression of follicular pustules [59]. Histopathology reveals this rare primary neutrophilic scarring alopecia [60]. Staphylococcus aureus is frequently detected in affected areas and considered pathogenic [61]. No definitive cure exists; treatment aims at stabilization with antibiotics/immunosuppressants [61]. Characteristic features include: Tufted hairs (multiple shafts per follicular opening), Perifollicular pustules/scaling, Late-stage follicular destruction [62] Deep fungal scalp infections may mimic FD (and vice versa), particularly in African Americans. Light microscopy and fungal culture are diagnostic essentials [63].

2.7.1 Staphylococcus aureus Infection

S. aureus colonizes 80% of FD patients' skin - the only bacterium consistently present in >2/3 cases [61]. During neutrophilic phases, follicular structural changes/immune dysfunction enable pathogenic S. aureus colonization, triggering neutrophilic inflammation that damages follicles [61]. S. aureus toxins complex with MHC proteins to stimulate T cells while evading immune detection. FD may result from abnormal host responses to post-infection toxin release [63].

2.7.2 Synergistic Effects of Polymicrobial Communities

Follicular biopsies from FD patients reveal the most prevalent bacterial genera include Staphylococcus, Cutibacterium, and Bacteroides. Staphylococcus accounts for 25.9% of the follicular microbiome in FD-affected biopsies, compared to only 6.6% in healthy follicles. Pathological follicles in FD patients exhibit distinct bacterial microbiota profiles versus healthy controls, suggesting this unique microbial signature may contribute to FD pathogenesis [63].

2.7.3 Propionibacterium acnes

Bacterial biofilms were observed in all patients and two of three control groups. These biofilms exclusively consisted of morphologically similar bacilli in both cohorts. While these bacilli were tentatively identified as Propionibacterium acnes, further validation is required. This study demonstrates the existence of bacterial biofilms in the infundibular-subinfundibular regions of human scalp follicles, detectable in both FD patients and controls, indicating their commensal existence with potential pathogenic transformation in FD.

2.7.4 Immunological Factors

As S. aureus may not be the sole pathogen, bacterial infection might only initiate rather than sustain the entire pathogenic process. Once immune-inflammatory cells are activated, follicular destruction persists even after pathogen clearance [64]. FD patients' intrinsic follicular, epidermal, or immune system alterations may predispose to localized microbial colonization (e.g., S. aureus) and subsequent folliculitis. However, whether specific bacteria are primary pathogens remains debatable.

During neutrophilic phases, hypothesized structural/immune dysfunction enables pathological S. aureus colonization, triggering neutrophilic inflammation that damages follicles. Chronic overstimulation by aberrant follicular microbiota may dysregulate monocyte cytokine production due to immune exhaustion. This impaired immunity complicates bacterial clearance, exacerbating disease progression.

3. Summary and Perspectives

Etiological analysis of these seven alopecias reveals that all ultimately converge on scalp/follicular immune imbalance. Whether through P. acnes/S. aureus-induced hyperimmunity or cellular immune dysregulation in AA due to genetic/lifestyle factors, the final common pathway involves immune attacks on follicles and arrested hair growth.

Thus, alopecias can be categorized into three types by etiology:

1. Androgen-induced immune activation (AGA, FFA): Minimal microbial involvement, primarily hormonal imbalance-driven immune dysregulation with significant genetic influence on hormone-related enzymes/receptors.
2. Microbe-induced immune activation (FD, LPP, TE): Chronic inflammation/immune stress from microbial dysbiosis/viral infection, where ROS pathway activation and IL-family cytokine dysregulation play pivotal roles.
3. Stress-associated multifactorial immune activation (CCCA, AA): Complex etiology with minimal microbial triggers, involving diverse cell types.

All pathways culminate in the "final common pathway" - immune imbalance disrupting follicular immune privilege.

For drug development, beyond disease-specific targets, universal therapeutic strategies should focus on: Androgen pathway modulation, Microbial homeostasis, Immune regulation targets. Emerging approaches like exosome-mediated immunomodulation and repurposing cancer immunotherapy targets (e.g., immune checkpoint molecules) may inspire localized scalp immunotherapies. With advancing research, clearer etiological mechanisms and shared pathways will undoubtedly emerge, guiding the development of broadly effective anti-alopecia therapies.

Table 1. Etiological Analysis of Several Diseases Leading to Hair Loss

	Immune Factors	Psychological Factors	Microbial Infection	Genetic Factors	Disease-Associated Factors	Possible Classification
Androgenic Alopecia (AGA)	+	+		+++	Coronary artery disease, benign prostatic hyperplasia, prostate cancer, psychological disorders (correlation)	I
Frontal Fibrosing Alopecia (FFA)	++			+	AGA, hormonal changes during menopause (direct factors)	I
Telogen Effluvium (TE)	+++		+++		High fever, malnutrition, wasting diseases, pro-inflammatory cytokines (direct factors)	II
Lichen Planopilaris (LPP)	+++			+++	Environmental pollution, heavy metal particles (correlation)	II
Central Centrifugal Cicatricial Alopecia (CCCA)	++	+	++	++	Leiomyoma, anxiety disorders, etc. (correlation)	III
Alopecia Areata (AA)	+++	+	++	+	Psychological disorders, poor living habits (correlation)	III
Folliculitis Decalvans (FD)	+++			+++	Bacterial, fungal infections (direct factors)	II

Note:"+" indicates the presence or involvement of the factor, "+++" indicates the highest level of involvement or strong direct correlation. The "Possible Classification" column categorizes the diseases based on the complexity and severity of their etiology.

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