

A biomarker-guided therapeutic map for alopecia: in vivo evidence and clinical implications

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Abstract

Alopecia is a multifactorial disorder involving disruption of immune, oxidative, apoptotic, and regenerative pathways in the hair follicle microenvironment. Despite therapeutic advances, clinical responses remain inconsistent, partly due to the limited understanding of underlying molecular targets. Integrating biomarker data may help clarify disease mechanisms and guide more targeted interventions. This review synthesizes in vivo animal evidence on six key biomarkers—tumor necrosis factor (TNF)- α , interleukin (IL)-6, malondialdehyde (MDA), caspase-3, vascular endothelial growth factor (VEGF), and β -catenin—and integrates their therapeutic implications into a biomarker-centered framework. A systematic search identified 15 animal studies (2014–2024) evaluating these biomarkers in alopecia models. TNF- α and IL-6 were generally elevated, indicating inflammation and immune-mediated follicular damage, whereas MDA reflected oxidative stress and caspase-3 activation marked apoptosis-driven regression. In contrast, VEGF and β -catenin were suppressed in alopecia but restored by effective interventions, supporting angiogenesis and Wnt-mediated regeneration. Various therapeutic approaches, including botanical extracts, bioactive compounds, sustained-release finasteride, microneedling, and nanoparticle-based delivery systems, were shown to reduce destructive biomarkers while enhancing regenerative pathways. Overall, alopecia can be conceptualized as an imbalance between degenerative and regenerative mechanisms, and a biomarker-guided therapeutic map may provide a useful translational framework for developing more precise and multitargeted treatment strategies.

Keywords: alopecia, angiogenesis, apoptosis, inflammation, Wnt signaling

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Introduction

Alopecia, particularly its subtypes androgenetic alopecia (AGA) and alopecia areata (AA), is a prevalent dermatological disorder with considerable global and regional impact. Epidemiological studies indicate that AGA is highly age dependent, affecting up to ~80% of Caucasian men and ~42% of women by late adulthood, although prevalence varies significantly across ethnicities (1). AA, in contrast, is an autoimmune-mediated form of nonscarring alopecia with a lifetime risk of approximately 2% worldwide, contributing substantially to psychosocial burden (2). In Indonesia, a multicenter dermatology series identified AGA (37.7%) and AA (18.2%) as the leading diagnoses of hair disorders, underscoring their local relevance (3). Both conditions are associated with impaired quality of life and increased psychological burden, including anxiety and depression (2, 4). Current therapeutic approaches remain limited: minoxidil and finasteride are the only U.S. Food and Drug Administration-approved drugs for AGA, but their efficacy is variable, adherence is often suboptimal, and side effects restrict long-term use (4). Adjunctive options, such as platelet-rich plasma (PRP), show signs of benefit but suffer from methodological heterogeneity and inconsistent outcomes (5, 6), whereas emerging agents for AA, including JAK inhibitors such as baricitinib, have demonstrated promising regrowth but require longer-term evaluation to establish durability and safety (7).

These therapeutic gaps highlight the need for greater insight into the molecular determinants of disease. Evidence implicates

several key biomolecules in alopecia pathophysiology: tumor necrosis factor (TNF)- α and interleukin (IL)-6 as well-established mediators of inflammation and immune privilege collapse (8, 9); malondialdehyde (MDA) as a marker of oxidative stress elevated in both AGA and AA (10, 11); caspase-3 as a key executioner of apoptosis involved in follicular regression (12); vascular endothelial growth factor (VEGF) as a central regulator of angiogenesis and follicular nourishment (13); and β -catenin as a critical component of Wnt signaling and follicle regeneration (14, 15). Importantly, TNF- α , IL-6, VEGF, and β -catenin are widely recognized as core regulators of inflammatory and regenerative pathways, whereas MDA and caspase-3 serve primarily as indicators of oxidative stress and apoptosis and are increasingly explored as supportive or emerging biomarkers in experimental models. Together, these six biomarkers were selected to provide an integrated representation of both destructive and regenerative mechanisms underlying alopecia and to support a biomarker-guided therapeutic framework.

Methods

Study design

This systematic review was conducted based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A systematic literature search was performed in PubMed, Scopus, ScienceDirect, and Google Scholar. The search strategy used a combination of keywords and Boolean operators,

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such as: (“Alopecia” OR “hair loss” OR “hair follicle”) AND (“TNF- α ” OR “IL-6” OR “MDA” OR “Caspase-3” OR “VEGF” OR “ β -catenin”) AND (“pathophysiology” OR “therapy” OR “treatment” OR “regeneration”).

Eligibility criteria

We included *in vivo* animal studies (any species or strain, including AA or AGA models) that 1) evaluated an intervention or exposure relevant to alopecia pathophysiology or therapy and 2) measured at least one of the prespecified biomarkers of interest—TNF- α , IL-6, MDA, caspase-3, VEGF, or β -catenin—in scalp skin, hair follicles, or other relevant tissue. Randomized and non-randomized controlled studies, as well as quasi-experimental designs, published in English between 2014 and 2024 were eligible. We excluded *in vitro* studies, human studies, studies involving pregnant animals or major comorbidities, case reports, narrative reviews, systematic reviews / meta-analyses, editorials, and studies that did not assess any of the prespecified biomarkers of interest.

Study selection

During the selection process, the titles and abstracts of potentially eligible studies were independently screened by three reviewers (AN, PD, and DGT). Full-text articles were subsequently evaluated for eligibility according to predefined inclusion and exclusion criteria. Any discrepancies among the reviewers were addressed through consensus-based discussion, and unresolved disagreements were adjudicated by a fourth reviewer (HK).

Risk of bias assessment

The risk of bias of the animal studies included was assessed using the SYRCLE risk of bias tool, a validated instrument specifically developed for animal intervention studies (16). This tool evaluates ten methodological domains, including sequence generation, baseline characteristics, allocation concealment, random housing, blinding of investigators and outcome assessors, incomplete outcome data, selective reporting, and other potential sources of

bias. The results of the risk-of-bias assessment were summarized using traffic-light plots and summary plots, indicating low, high, or unclear risk for each domain.

Data extraction and analysis

The data analysis in this systematic review involved summarizing the findings of the studies included using a narrative and thematic synthesis. Data were evaluated qualitatively to identify consistent patterns in interventions and biomarker outcomes. Because no meta-analysis was conducted, publication bias, heterogeneity, and sensitivity analyses were not applicable. Given the preclinical nature of the studies, no data transformation was required. Results were organized according to the prespecified biomarker domains; namely, inflammatory (TNF- α and IL-6), oxidative (MDA), apoptotic (caspase-3), and regenerative (VEGF and β -catenin) pathways. The certainty of evidence was not formally assessed using tools such as GRADE, given the heterogeneity and preclinical nature of the animal studies included.

Results

The PRISMA diagram shown in Figure 1 illustrates the study selection process. A total of 3,581 studies were identified from four databases: PubMed (480 records), ScienceDirect (1,225 records), Scopus (916 records), and Google Scholar (960 records). Before screening, 565 duplicate records were removed. After screening titles and abstracts, 2,700 studies were excluded, comprising 2,200 studies not specifically addressing alopecia therapy and biomolecular aspects, 34 articles published in languages other than English, and 466 review articles, books, case reports, or guidelines. This process left 316 full-text articles for eligibility assessment. Of these, 301 were further excluded: 115 studies were conducted in humans, 92 were *in vitro* experiments, and 94 did not provide eligible *in vivo* outcome data for the prespecified biomarkers or otherwise failed to meet the inclusion criteria. Ultimately, 15 studies met all the inclusion criteria and were incorporated into the final review.

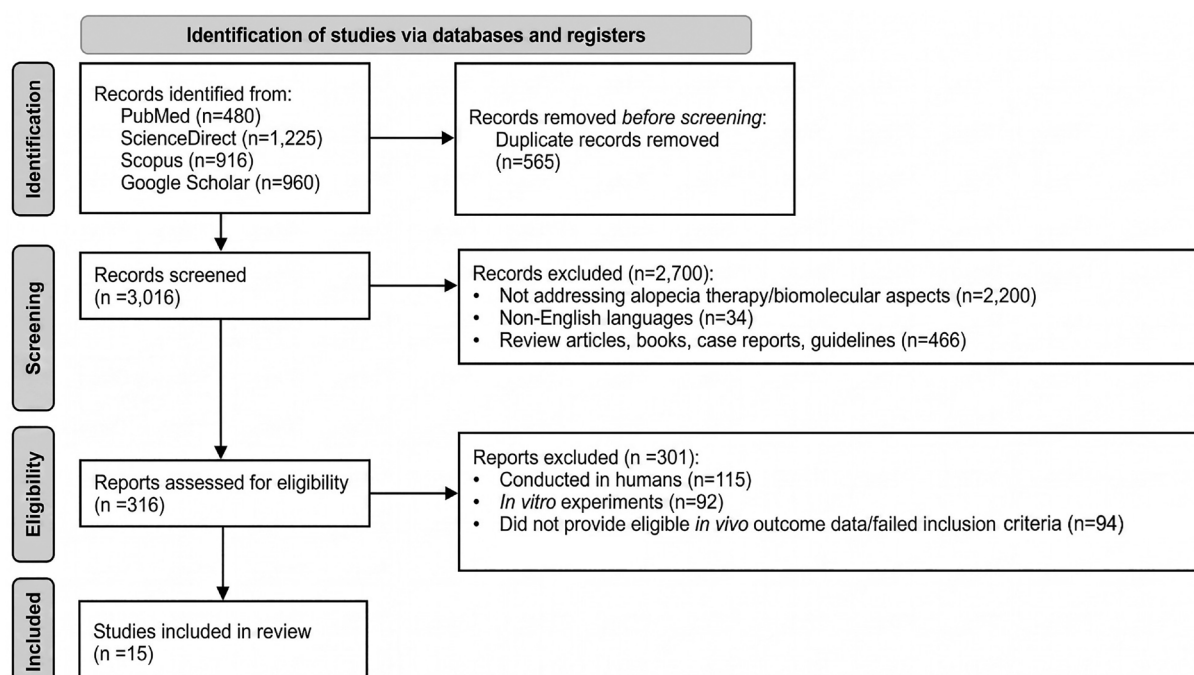


Figure 1 | PRISMA flowchart.

Risk of bias assessment

We evaluated the animal studies included using SYRCLE’s risk-of-bias tool. The instrument covers ten domains, each judged as yes, no, or unclear, corresponding to low, high, or unclear risk of bias, respectively. Each domain was appraised and interpreted independently (16). Figure 2 summarizes the judgments in a traffic-light plot.

Study characteristics

A total of 15 in vivo studies met the eligibility criteria and were included, encompassing both AGA and AA models (Table 1). The majority (*n* = 9) employed C57BL/6 mice or related sub-strains, and two utilized C3H/HeJ mice, a strain particularly relevant for AA due to its autoimmune susceptibility. Sample sizes per group generally ranged from six to 10 animals, with treatment durations between 4 and 12 weeks, depending on the experimental design.

The studies included investigated a wide range of therapeutic approaches. These comprised natural extracts (*Paeonia lactiflora*, *Poria cocos*, oleanolic acid, *Rosa rugosa*, Xuefu Zhuyu decoction, and Shen Bai hair-growing decoction), bioactive compounds (amygdalin and policosanol), and advanced delivery systems such as poly(lactic-co-glycolic acid) (PLGA)-encapsulated fin-

asteride microspheres, marine collagen peptide-incorporated microneedles, and safflower oil body nanoparticles loaded with FGF10. In addition, one study explored microneedle-induced microwounding as a purely physical intervention. Together, these models provide complementary perspectives on how pharmacological, botanical, and mechanical strategies can modulate biomarker pathways and promote hair regrowth. Overall, the studies included predominantly utilized murine models, particularly C57BL/6 and C3H/HeJ strains, reflecting their established relevance in alopecia research. Most studies focused on AGA models, with a smaller subset investigating AA, highlighting a greater representation of androgen-driven pathophysiology in the current evidence base.

Discussion

Inflammatory pathways (TNF-α, IL-6)

Across animal models, the pro-inflammatory cytokines TNF-α and IL-6 generally emerged as key drivers of follicular pathology. Both the AGA and AA models showed elevated levels of these cytokines compared to healthy controls (17, 18, 25, 26). For example, He et al. observed marked upregulation of TNF-α and IL-6 in a C3H/HeJ mouse model of AA, coinciding with immune cell infiltration and

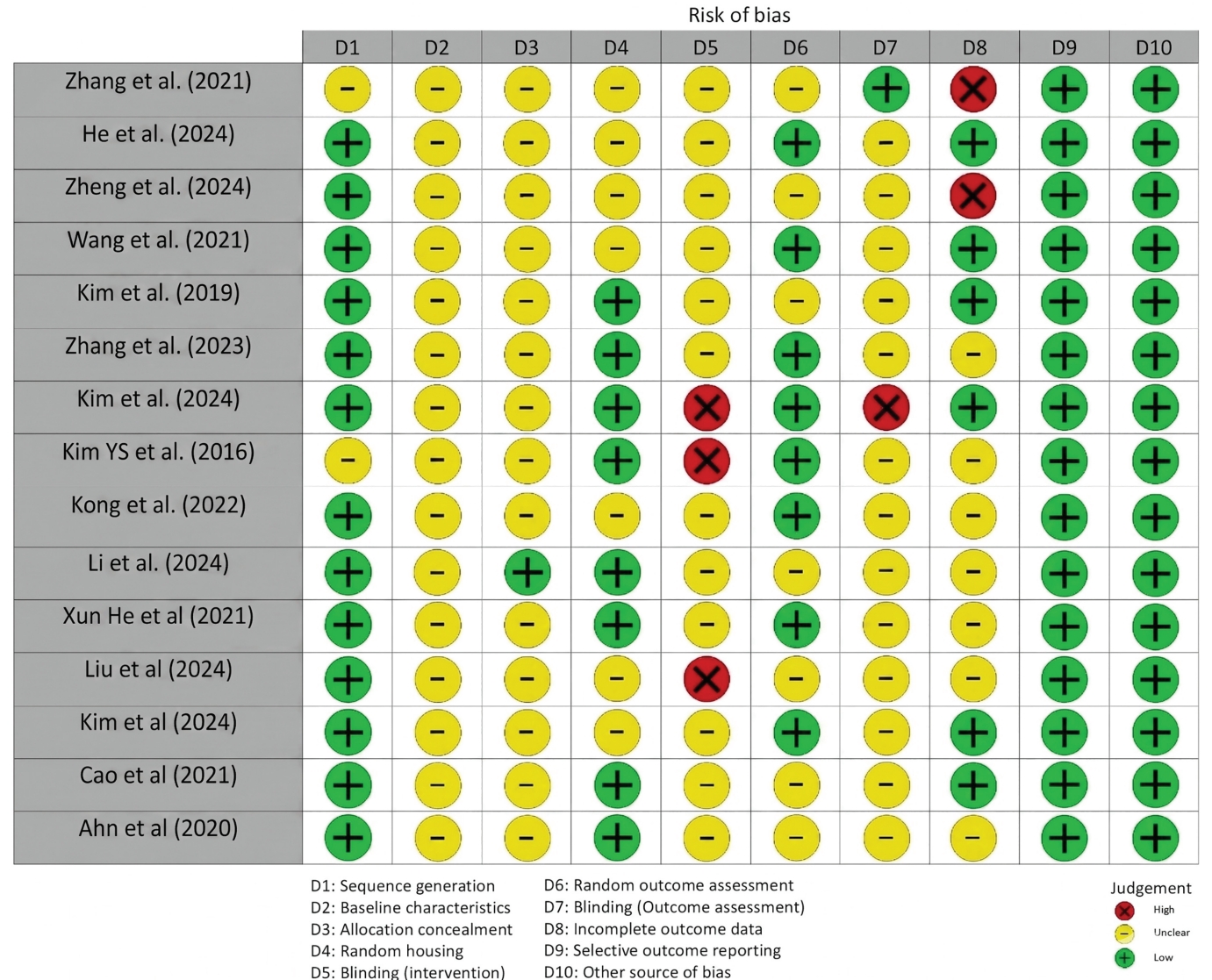


Figure 2 | Summary plot of the SYRCLE risk-of-bias assessment for the animal studies included.

extensive follicular damage (26). Similarly, in an androgen-driven context, Zhang et al. reported substantially higher TNF- α and IL-6 expression in the skin of testosterone-treated AGA mice than in normal controls (17). These findings indicate that both AA and AGA involve an inflammatory microenvironment that is hostile to hair follicles. TNF- α is known to disrupt hair follicle immune privilege and trigger local tissue injury, thereby promoting catagen entry and follicular miniaturization (17, 26). IL-6 may act on follicular keratinocytes and dermal papilla cells via JAK/STAT signaling, promoting Th1/Th17 immune responses and suppressing hair growth (26). These animal findings are consistent with clinical evidence showing elevated serum TNF- α and IL-6 in patients with AA (8), supporting the central role of these cytokines in alopecia pathogenesis. In AGA, although the primary driver is hormonal, perifollicular “microinflammation,” potentially mediated by IL-6 and related cytokines, is increasingly recognized as contributing to disease progression (17). Taken together, TNF- α and IL-6 appear to function as pivotal inflammatory mediators linking diverse alopecia subtypes to a shared pathway of follicular injury.

Therapeutic interventions generally reduced TNF- α and IL-6, confirming their relevance as modifiable disease drivers. Multiple treatments associated with hair regrowth also suppressed one or both of these inflammatory markers. In an AA model, amygdalin significantly reduced elevated TNF- α and IL-6 levels, accompanied by decreased CD8 $^{+}$ T-cell infiltration and restoration of regulatory T-cell activity (18). Likewise, in AGA models, botanical extracts showed anti-cytokine effects: aqueous *Paeonia lactiflora* extract normalized TNF- α in balding mice (17), whereas Shen Bai hair growing decoction markedly lowered TNF- α and IL-6 in testosterone-treated mice (25). Novel follicle-targeted delivery systems showed similar results; for example, safflower oil body nanoparticles carrying FGF10 significantly inhibited dihydrotestosterone (DHT)-induced TNF- α /IL-6 overproduction in AGA mice while accelerating hair regrowth (24). Notably, across the studies included, no effective therapy increased these cytokines; the overall trend was generally toward cytokine reduction with successful treatment. In several studies, the degree of TNF- α /IL-6 suppression appeared to parallel the extent of hair recovery (18, 26). Some interventions showed more selective effects; for example, oleanolic acid reduced TNF- α and transforming growth factor (TGF)- β 1 but had a minimal impact on IL-6 (21), suggesting that certain treatments may resolve inflammation through partially distinct mechanisms. Overall, suppression of the TNF- α /IL-6 axis appears to be beneficial for follicular preservation. Translationally, these findings support inflammation as a meaningful therapeutic target. Clinically established agents that modulate these pathways, including JAK inhibitors such as baricitinib and corticosteroid-based therapies, further reinforce the relevance of inflammatory signaling in alopecia management. However, systemic cytokine blockade carries immunologic risks, underscoring the importance of localized or upstream modulation within the scalp. Collectively, these findings support TNF- α and IL-6 as potentially useful biomarkers of inflammatory disease activity and as promising therapeutic targets.

Oxidative stress (MDA)

Oxidative stress is another prominent pathological component in alopecia models, most commonly reflected by elevated MDA levels in the scalp. As a reactive end-product of lipid peroxidation, MDA was generally higher in alopecic animals than in controls in stud-

ies that measured it (31). For example, Zheng et al. demonstrated that testosterone exposure significantly increased scalp MDA in an AGA mouse model, indicating excessive reactive oxygen species (ROS) production and associated lipid peroxidation during hair loss (31). This oxidative burden may directly injure follicular cells by damaging DNA, proteins, and cell membranes, thereby accelerating apoptosis and premature catagen entry. These findings parallel clinical observations in both AGA and AA, in which oxidative imbalance and elevated MDA or related oxidative markers have also been reported (10, 11). Mechanistically, androgen-driven alopecia may promote ROS generation through androgen metabolism and local inflammation, whereas chronic inflammation in AA may similarly intensify oxidative stress within the follicular microenvironment. Thus, MDA appears to function as a broad indicator linking metabolic and inflammatory stress to alopecia pathophysiology.

Therapies that reduced oxidative stress were generally associated with improved hair growth. Although fewer studies quantified MDA than inflammatory cytokines, those that did generally found that successful interventions lowered MDA levels. For instance, Zheng et al. showed that collagen peptides delivered through microneedles reduced scalp MDA in AGA mice, correlating with earlier anagen induction and denser regrowth (31). By limiting lipid peroxidation and neutralizing free radicals, such interventions may create a more favorable environment for follicular recovery. Even in studies that did not directly measure MDA, some effective agents likely exerted antioxidant effects; for example, oleanolic acid has demonstrated free radical-scavenging capacity in vitro, suggesting that part of its benefit may derive from reducing oxidative burden (21). Because inflammation and oxidative stress are closely linked, anti-inflammatory therapies may also mitigate ROS generation indirectly. A limitation of the current evidence is that relatively few studies assessed oxidative markers, making it difficult to determine which therapies most effectively target ROS-related damage. Future preclinical work should incorporate additional oxidative indices, such as superoxide dismutase and glutathione, to better characterize antioxidant mechanisms. Nevertheless, the repeated association between reduced MDA levels and improved regrowth supports oxidative stress as an important contributor to alopecia. Translationally, these findings support incorporation of antioxidant strategies into treatment paradigms, whether through dietary interventions, topical therapies, or advanced delivery systems that protect follicles from oxidative injury and complement anti-inflammatory or anti-androgenic approaches.

Apoptosis (caspase-3)

Programmed cell death of follicular cells is a central mechanism of hair loss, with caspase-3 acting as a key apoptotic executioner. Several animal studies showed that excessive caspase-3 activation accompanies follicular regression. In AGA models, androgen (DHT)-induced stress appears to trigger intrinsic apoptosis, prematurely terminating anagen. Wang et al. observed upregulated caspase-3, Bax, and caspase-9 in untreated AGA mice, correlating with accelerated catagen transition and hair loss (19). Similarly, Kim et al. reported increased cleaved caspase-3 and poly ADP-ribose polymerase (PARP) in testosterone-driven alopecia, indicating active apoptosis and progressive follicular miniaturization (20). These findings support the concept that hair-cycle regression is, at least in part, apoptosis dependent because matrix and outer

root sheath cells undergo programmed death, resulting in follicular shrinkage and hair thinning. Although AA models more often focused on immune-mediated mechanisms, apoptosis likely also contributes to follicular loss in that setting. Human studies further support this view, showing increased caspase-3 expression in balding scalp biopsies that was normalized by finasteride treatment (32). Overall, excessive caspase-3 activity appears to represent a convergence point at which androgens, oxidative stress, and inflammation translate into irreversible follicular injury.

Treatments that preserved hair follicles generally suppressed caspase-3 activation, supporting apoptosis inhibition as an important therapeutic strategy. Most interventions associated with hair regrowth also reduced caspase-3 expression or activity. For example, topical policosanol improved hair density in AGA mice while downregulating caspase-3 and caspase-9 and upregulating Bcl-2, thereby shifting the Bax/Bcl-2 balance toward cell survival and prolonging anagen (19). Similarly, a single injection of finasteride-loaded polymer microspheres achieved regrowth comparable to that of daily oral finasteride, with both approaches reducing follicular caspase-3 (20). Multi-herb formulations such as Shen Bai hair growing decoction also decreased caspase-3 expression in parallel with anti-inflammatory effects (25). No effective therapy increased caspase-3, reinforcing the idea that limiting follicular apoptosis is essential for hair preservation. Although some AA studies did not measure apoptosis directly, attenuation of immune-mediated injury likely reduced downstream apoptotic cell loss. Despite variation in biomarker assessment methods, all relevant studies showed reduced caspase-3 activity with treatment. Collectively, these findings establish caspase-3 as a useful marker of follicular viability: when its activity declines, follicles are more likely to remain in the growth phase. Translationally, these results support strategies that enhance pro-survival signaling, such as Bcl-2 upregulation or modulation of related apoptotic pathways, as potential adjuncts to current therapies. Because complete apoptosis blockade would be biologically undesirable, targeted protection of follicular cells is a more realistic therapeutic goal. Indeed, clinically used agents such as finasteride appear to exert part of their benefit through reduction of follicular apoptosis (32). Future therapies may build on this principle by promoting a scalp microenvironment that protects growth-supporting cells from premature death.

Regenerative mechanisms (VEGF, β -catenin)

Although alopecia involves several destructive processes, the studies reviewed also highlight opposing regenerative pathways, particularly those involving VEGF and β -catenin, which are critical for follicular growth and cycling. Adequate vascularization is essential for maintaining anagen, and VEGF is the principal pro-angiogenic factor implicated in hair follicle biology. In alopecia models, reduced VEGF was associated with poor growth, whereas therapies that restored VEGF promoted regeneration. Untreated AGA mice exhibited lower VEGF levels and fewer perifollicular capillaries, contributing to follicular miniaturization (19, 31). Conversely, nearly all effective treatments increased VEGF expression: microneedle-delivered collagen peptides elevated VEGF and CD31, indicating neovascularization accompanied by denser regrowth (31); policosanol increased VEGF and epidermal growth factor (EGF) in the scalp (19); and oleanolic acid and *Rosa rugosa* extract dose-dependently upregulated VEGF, enhancing follicular proliferation and thickness (21, 22). Even microneedling alone in-

creased local VEGF and improved follicle maturation (23). Collectively, these findings identify VEGF as a central mediator of follicular recovery, with its restoration by drugs, natural compounds, or physical stimulation generally associated with improved hair growth. Studies using minoxidil as a control further support this mechanism because minoxidil stimulates VEGF through hypoxia-inducible factor (HIF)-1 α -related signaling, enlarges follicles, and prolongs anagen (13, 22). None of the successful interventions inhibited VEGF; at most, some simply did not assess it. Although VEGF data in AA models remain limited, reduction of inflammatory burden may secondarily improve local perfusion. Clinically, these observations reinforce the value of pro-angiogenic strategies in alopecia treatment. Established therapies such as minoxidil and platelet-rich plasma already exploit VEGF-related mechanisms, and future approaches may benefit from growth factor delivery systems or biomaterials that more effectively enhance scalp vascularization.

The Wnt/ β -catenin pathway is fundamental for hair follicle stem cell activation and cycling, and the studies reviewed reaffirm its central role in alopecia pathogenesis and treatment (14). β -catenin acts as a molecular switch for anagen initiation, and its suppression was generally associated with hair loss, whereas effective therapies repeatedly reactivated this pathway. In AGA models, DHT-induced Wnt antagonists such as DKK1 suppressed β -catenin; Zhang et al. found elevated DKK1 and reduced β -catenin in follicles, both of which were reversed by *Paeonia lactiflora* extract, with corresponding hair regrowth (17). Similarly, finasteride and finasteride-loaded microspheres restored Wnt3a and β -catenin expression, preserving active follicles (20). Other interventions converged on the same regenerative pathway: *Rosa rugosa* extract enhanced nuclear β -catenin in dermal papilla cells (22), oleanolic acid increased β -catenin in matrix cells (21), microneedling elevated Wnt3a, Wnt10b, and β -catenin expression (23), cardamomin liposomes activated both Wnt/ β -catenin and sonic hedgehog signaling (27), and *Justicia procumbens* extract stabilized β -catenin through GSK-3 β inhibition, thereby inducing downstream targets such as cyclin D1 and VEGF (28). Even advanced nanocarrier systems, such as finasteride-loaded lipid microneedles, suppressed DKK1 while enhancing β -catenin and growth-related signaling (29). Across the studies included, β -catenin upregulation—either directly or through inhibition of upstream Wnt suppressors—was a recurring hallmark of successful hair regrowth.

Translationally, these findings support Wnt/ β -catenin as a final common regenerative pathway through which diverse therapies promote follicular recovery. This makes it an attractive therapeutic target because reactivation of Wnt signaling may help revive dormant follicles and stimulate new growth (14). However, systemic Wnt activation raises oncogenic concerns, underscoring the need for localized or transient stimulation. Approaches such as microneedling, follicle-targeted Wnt agonists, and topical modulators such as valproic acid or KY19382 may offer more controlled activation and are of particular translational interest. Overall, the regenerative evidence suggests that effective alopecia management requires not only suppression of destructive pathways—namely inflammation, oxidative stress, and apoptosis—but also active restoration of regenerative signaling through VEGF and Wnt/ β -catenin.

The current evidence is limited by the relatively small number of in vivo studies, the heterogeneity of animal models, and variability in biomarker assessment, all of which constrain gen-

eralizability and preclude meta-analysis. Importantly, the available data are predominantly derived from murine models, with a greater representation of AGA than AA. This imbalance may limit the applicability of the findings across all alopecia subtypes, particularly those driven primarily by autoimmune mechanisms. In addition, this review excluded in vitro and clinical studies, and publication bias was not formally assessed because of the qualitative nature of the synthesis.

From a therapeutic perspective, the studies reviewed suggest that current and emerging alopecia treatments can be mapped according to the biomarker pathways they modulate. Clinically established therapies already target several of these pathways: JAK inhibitors and corticosteroid-based therapies primarily suppress inflammatory signaling, minoxidil enhances angiogenesis through VEGF-related mechanisms, and finasteride reduces androgen-driven apoptosis while indirectly supporting regenerative pathways such as Wnt/ β -catenin. Although these agents were not uniformly evaluated across the animal studies included, their established clinical use strengthens the translational relevance of the biomarker-guided framework proposed here. In parallel, several natural products and bioactive compounds reviewed in this study, including *Paeonia lactiflora*, oleanolic acid, policosanol, and *Rosa rugosa* extract, demonstrated multimodal activity by suppressing inflammatory, oxidative, and apoptotic markers while enhancing VEGF and β -catenin. This convergence suggests that effective alopecia therapies may not act through a single pathway, but rather through coordinated modulation of destructive and regenerative mechanisms. Accordingly, a biomarker-guided therapeutic map may help classify existing therapies, identify mechanistic gaps, and support development of combination or localized strategies that more precisely target follicular pathology. To further clarify

these therapeutic-pathway relationships, Table 2 summarizes representative therapies and their reported effects on inflammatory, oxidative, apoptotic, angiogenic, and Wnt-related biomarkers.

Taken together, these findings frame alopecia as a disorder of imbalance between destructive mechanisms (inflammation, oxidative stress, and apoptosis) and regenerative mechanisms (VEGF- and Wnt/ β -catenin-mediated repair). The reviewed therapies appear to restore this balance through combined anti-inflammatory, antioxidant, anti-apoptotic, and pro-regenerative effects. This framework also helps position current and emerging therapies according to the molecular pathways they influence, thereby strengthening the translational value of biomarker-based treatment selection. Overall, the proposed biomarker-centered therapeutic map highlights opportunities for more precise, multi-targeted, and localized strategies in alopecia management.

Conclusions

This review demonstrates that alopecia in animal models reflects a disruption between inflammatory, oxidative, and apoptotic processes versus angiogenic and Wnt-driven regenerative mechanisms. Therapeutic interventions that simultaneously suppress destructive biomarkers (TNF- α , IL-6, MDA, and caspase-3) and enhance regenerative ones (VEGF and β -catenin) appear to offer the greatest therapeutic potential for restoring follicular health. Current and emerging therapies, including anti-inflammatory agents, anti-androgenic strategies, pro-angiogenic interventions, and Wnt-targeting approaches, can be interpreted within this biomarker-guided framework. Framing these findings as a biomarker-centered therapeutic map provides a translational framework for developing multi-targeted strategies in alopecia management.

Table 1 | Summary of in vivo studies included and biomarker-related findings in alopecia models.

Source	Sample (number, population)	Aim	Key findings
Zhang et al. (2021) (17)	Species: C57BL/6 mice Groups: six – Normal-con (no AGA induction, ethanol only) – AGA-con (testosterone-induced, cellulose diet) – AGA-PL (0.1% PL extract) – AGA-PC (0.1% PC extract) – AGA-MIX (0.05% PL + 0.05% PC) – AGA-positive (2% finasteride topical)	To evaluate the efficacy of PL and PC in AGA treatment and their molecular mechanism in network pharmacology and an established AGA animal model	PL and PL+PC improved hair growth, restored follicle density, reduced testosterone, and suppressed inflammatory cytokines. PL was more effective than PC alone.
He et al. (2024) (18)	Species: C3H/HeJ female mice Groups: four – Control – AA – Ruxolitinib (60 mg/kg) – Amygdalin (60, 90, 120 mg/kg)	To investigate the therapeutic effects of amygdalin on AA in C3H/HeJ mice, focusing on its molecular mechanism in regulating inflammation and immune response through the JAK2/STAT3 pathway	Amygdalin improved hair growth, reduced IL-6, TNF- α , and IFN- γ , improved CD4 $^{+}$ /Treg cells, reduced CD8 $^{+}$, and suppressed JAK2/STAT3. Conclusion: amygdalin inhibits inflammation and modulates immunity in AA via the JAK2/STAT3 pathway; it is a potential therapeutic candidate.
Zheng et al. (2024) (31)	Species: male C57BL/6 mice Number of species: one (mouse) Groups: six (n = 6 each) – Topical GSCPs – Blank microneedles (MNs) – GSCPs-MNs – Blank control (healthy) – AGA (negative control, testosterone-induced) – Minoxidil (positive control)	To evaluate the efficacy of GSCP-incorporated dissolving MNs in promoting hair regrowth in AGA by reducing inflammation and oxidative stress, and enhancing vascularization and follicle regeneration	The GSCPs-MNs group showed earlier and denser hair regrowth, larger hair diameter, enhanced collagen deposition, reduced lipid peroxidation (MDA), and increased VEGF and CD31 expression. Inflammatory cytokines (TNF- α , IL-1 β) were significantly reduced compared to the AGA and topical GSCPs groups. Blank MNs alone also promoted some hair regrowth, suggesting a synergistic effect of microneedling and collagen peptides. Conclusion: GSCPs-MNs effectively alleviated AGA symptoms in mice by promoting angiogenesis, reducing oxidative stress, and modulating inflammatory markers. The treatment prolonged the anagen phase and improved hair shaft quality, demonstrating potential as a novel therapeutic and cosmetic intervention for hair loss.
Wang et al. (2021) (19)	Species: Kunming mice (male, 4 weeks old) Groups: six (n = 8 each) – Control (untreated, CK) – Model (testosterone propionate only) – Positive control (testosterone + 2% finasteride) – Testosterone + 0.5% policosanol – Testosterone + 1% policosanol – Testosterone + 2% policosanol	To elucidate the mechanism of policosanol from Chinese wax in preventing AGA by regulating abnormal hormone levels, modulating cytokine expression, and suppressing premature hair follicle regression	Policosanol reduced testosterone and the T/E2 ratio, increased VEGF & EGF, enhanced ALP, stimulated DPC proliferation, downregulated TGF- β 2, Bax, caspase-9, and caspase-3, and upregulated Bcl-2. Conclusion: policosanol prevents AGA by restoring hormone balance, enhancing growth factor expression, protecting DPCs from apoptosis, and delaying follicle regression.
Kim et al. (2019) (20)	Species: male C57BL/6 mice Number of species: one (mouse) Groups: four – Control (no testosterone, no finasteride) – Testosterone + placebo microspheres – Testosterone + oral finasteride – Testosterone + finasteride-loaded microspheres	To confirm the efficacy of a single injection of finasteride-loaded microspheres on the alleviation of hair loss through an in vivo experiment, by comparing its effects with those of daily orally applied finasteride treatment in mice	Finasteride-loaded microspheres significantly improved hair regrowth in testosterone-induced alopecia mice, with 93.3% showing regrowth compared to 86.7% in the oral finasteride group. Both treatments enhanced follicle number, length, diameter, and anagen/telogen ratio. At a molecular level, β -catenin and Wnt3a signaling were upregulated, apoptotic markers (caspase-3, Bax, TGF- β 2, cleaved-PARP) were suppressed, and Bcl-2 expression was increased. Serum DHT levels were markedly reduced after week 6 in both groups. Conclusion: a single subcutaneous dose of finasteride-loaded PLGA microspheres produced hair growth and molecular effects equivalent to daily oral finasteride, indicating its promise as a sustained-release systemic therapy for androgenic alopecia.

Table 1 | Continued.

Source	Sample (number, population)	Aim	Key findings
Zhang et al. (2023) (21)	Species: male C57 mice Number of species: one (mouse) Groups: six ($n = 10$ each) – Control (blank emulsion) – Testosterone (blank emulsion + testosterone) – Minoxidil (5%) – OA-H (1% OA) – OA-M (0.5% OA) – OA-L (0.05% OA)	To evaluate the effects of OA on hair growth in testosterone-induced AGA in mice, focusing on inflammatory cytokine regulation and hair follicle growth mechanisms	OA significantly enhanced hair growth and promoted progression into the anagen phase. OA decreased inflammatory cytokines TNF- α and TGF- β 1 (not IL-6). OA increased VEGF levels and β -catenin expression, stimulating follicle proliferation. OA downregulated DHT and 5 α -reductase, key contributors to AGA. Antioxidant and antibacterial activities of OA were also confirmed. Conclusion: OA promotes hair growth through Wnt/ β -catenin pathway activation, angiogenesis (via VEGF), and suppression of inflammatory mediators (TNF- α , TGF- β 1), supporting its potential as a therapeutic agent for alopecia.
Kim et al. (2024) (22)	Species: male C57BL/6J mice Groups: five – Control – DHT – RWE 25 mg/kg – RWE 100 mg/kg – Bicalutamide	To assess the inhibitory effects and mechanisms of RWE in a DHT-induced alopecia mouse model	RWE stimulated hair regrowth, increased thickness, enhanced VEGF, EGF, IGF-1, reduced IL-6 and DKK-1, and activated the β -catenin pathway.
Kim et al. (2016) (23)	Female C57BL/6 mice (7 weeks old) divided into groups based on microneedle length (0.15, 0.25, 0.5, 1.0 mm) and rolling cycles (3, 6, 10, 13), with a control group receiving rollers without microneedles	To examine whether repeated microneedle-induced microwounds could promote hair growth and influence expression of hair-growth-related genes in mice	0.25 mm / 10 cycles increased Wnt3a and Wnt10b, and 0.5 mm / 10 cycles significantly upregulated Wnt3a, β -catenin, VEGF, and Wnt10b. Conclusion: repeated microneedle stimulation enhanced hair growth in mice via activation of the Wnt/ β -catenin pathway and VEGF signaling, suggesting potential as a therapeutic strategy for alopecia.
Kong et al. (2022) (24)	72 C57BL/6 mice (six groups); 27 ICR mice (three groups); 30 guinea pigs (three groups); 20 C57BL/6 mice (toxicity test)	To evaluate the therapeutic potential of SOB-hFGF10 for targeting hair follicles, reducing microinflammation, and accelerating hair regeneration in AGA	SOB-hFGF10 accelerated hair regeneration in AGA mice, inhibited DHT-induced TNF- α , IL-1 β , IL-6 overproduction, promoted DPC proliferation, and showed similar efficacy to minoxidil but without toxicity. Conclusion: SOB-hFGF10 is a safe, natural, and effective topical system for AGA treatment with strong anti-inflammatory and follicle-targeting properties.
Li et al. (2024) (25)	Species: male C57BL/6 mice (6 weeks old, 18–22 g) Groups: five – Control (did not receive testosterone) – Model (only testosterone) – TSBHGD low-dose (testosterone + TSBHGD low-dose) – TSBHGD high-dose (testosterone + TSBHGD high-dose treated) – Positive (testosterone + minoxidil treated)	To investigate the efficacy and underlying mechanism of TSBHGD against AGA using a testosterone-induced mouse model	SBHGD showed remarkable hair growth-promoting activity, suggesting that TSBHGD exerts a pronounced antialopecia effect in the AGA mouse model. The results suggest that the development of AGA is accompanied by increased levels of inflammatory factors and topical application of TSBHGD can inhibit TNF- α and IL-6 levels. The level of caspase-3 was remarkably decreased in all dose groups and the positive group.
Xun He et al. (2021) (26)	Species: Female C3H/HeJ mice (6–8 weeks old). Groups: five ($n = 6$ each) – Control – CUMS (AA model) – XZD low dose (6.5 g/kg/day) – Medium dose (13.0 g/kg/day) – High dose (26.0 g/kg/day)	To determine whether XZD promotes hair regeneration in AA through anti-inflammatory mechanisms	XZD significantly promoted hair regeneration in AA mice by increasing hair follicle numbers and reducing skin lesions. It downregulated IL-6, IL-1 β , TNF- α , and osteopontin at both the protein and gene levels. XZD ameliorates CUMS-induced AA by suppressing pro-inflammatory cytokines and enhancing hair follicle regeneration.

Table 1 | Continued.

Source	Sample (number, population)	Aim	Key findings
Liu et al. (2024) (27)	C57BL/6 mice (5–7 weeks old, testosterone-induced AGA model, $n = 6$ per group), New Zealand white rabbits (skin irritation test, $n = 4$)	To develop and evaluate a CAR@Lip formulation (gel) for improving skin penetration, follicular delivery, and therapeutic efficacy against AGA	CAR@Lip treatment upregulated VEGF, IGF-1, KGF, β -catenin, and SHH/Gli signaling, while downregulating TGF- β , leading to enhanced angiogenesis, follicular regeneration, higher anagen/telogen ratio, and improved hair regrowth compared with controls and minoxidil. CAR@Lips restore balance between inhibitory (TGF- β) and regenerative (VEGF, IGF-1, KGF, and β -catenin) pathways, supporting their potential as a safe and effective therapeutic option for alopecia.
Kim et al. (2024) (28)	B6CBAF1/J mice, testosterone-induced androgenic alopecia (AGA) model	To test JPAE against AGA via Wnt/ β -catenin pathway activation	JPAE increased β -catenin, p-GSK-3 β , VEGF, and cyclin D1, decreased Dkk-1, and promoted follicular proliferation and hair regeneration. Conclusion: JPAE prevents hair loss by restoring Wnt/ β -catenin signaling and enhancing regenerative biomarkers, indicating potential as a natural anti-AGA therapy.
Cao et al. (2021) (29)	C57BL/6 mice (male, 8 weeks old, testosterone-induced AGA model, $n = 6$ per group)	To develop a FIN-NLC-MN for finasteride delivery to hair follicles, improving follicular targeting, drug uptake, and therapeutic efficacy in AGA	FIN-NLC-MN downregulated SRD5A2, DKK-1, and TGF- β 1, upregulated β -catenin, IGF-1, and VEGF, accelerated anagen entry, restored follicle morphology, and promoted stronger regrowth in comparison to minoxidil and FIN suspension. Conclusion: FIN-NLC-MN restores balance between inhibitory and regenerative biomarkers, offering a potent and targeted approach for AGA therapy.
Ahn et al. (2020) (30)	14 male C57BL/6 mice (6 weeks old, telogen phase), divided into two groups ($n = 7$ each: control vs. treatment), topical treatment applied for 17 days	To evaluate effect of PEaT on hair growth and mechanisms	PEaT increased IGF-1, VEGF, Ki-67, wnt3, and β -catenin expression, accelerated anagen entry, and promoted follicular proliferation and hair regrowth. PEaT enhances regenerative biomarker pathways, supporting its role as a natural hair growth promoter.

AA = alopecia areata, AGA = androgenetic alopecia, ALP = alkaline phosphatase, Bax = Bcl-2-associated X protein, Bcl-2 = B-cell lymphoma 2, CAR@Lip = cardamonin-loaded liposome, DHT = dihydrotestosterone, DKK-1 = dickkopf-1, DPC = dermal papilla cells, EGF = epidermal growth factor, FIN-NLC-MN = finasteride-loaded nanostructured lipid carrier microneedle, GSCP = globefish skin collagen peptide, GSCPs-MN = collagen peptide-loaded microneedle, IFN- γ = interferon-gamma, IGF-1 = insulin-like growth factor-1, IL-6 = interleukin-6, JAK/STAT = Janus kinase / signal transducer and activator of transcription, JPAE = aqueous *Justicia procumbens* extract, KGF = keratinocyte growth factor, MDA = malondialdehyde, MN = microneedle, OA = oleanolic acid, PARP = poly (ADP-ribose) polymerase, PC = *Poria cocos*, PEaT = *Platycladus orientalis* extract with α -terpineol, PL = *Paeonia lactiflora*, RWE = *Rosa rugosa* water extract, SOB-hFGF10 = safflower oil body-human fibroblast growth factor 10, TGF- β = transforming growth factor-beta, TNF- α = tumor necrosis factor-alpha, TSBHGD = topical Shen Bai hair growing decoction, VEGF = vascular endothelial growth factor, XZD = Xuefu Zhuyu decoction.

Table 2 | Representative alopecia therapies and their reported effects on key pathogenic biomarker pathways.

Therapy/ intervention	Main therapeutic category	Targeted biomarker/pathway(s)	Reported/pathophysiologic effect
JAK inhibitors (e.g., baricitinib)	Anti-inflammatory / immunomodulatory	IL-6-related signaling, JAK/STAT pathway, inflammatory cascade	Suppress immune-mediated follicular injury and reduce inflammatory signaling, especially relevant in AA
Corticosteroids	Anti-inflammatory / immunosuppressive	TNF- α , IL-6, inflammatory infiltrates	Reduce perifollicular inflammation and immune activation
Minoxidil	Pro-regenerative / pro-angiogenic	VEGF, perifollicular vascularization	Enhances angiogenesis, prolongs anagen, improves follicular nourishment
Finasteride	Anti-androgen / anti-apoptotic / regenerative support	Caspase-3, androgen-driven follicular stress, indirect Wnt support	Reduces follicular miniaturization and apoptosis, supports maintenance of active follicles
Microneedling	Physical regenerative stimulation	VEGF, Wnt/ β -catenin	Promotes wound-healing signals, angiogenesis, and follicular regeneration
<i>Paeonia lactiflora</i> extract	Botanical multimodal therapy	TNF- α , β -catenin	Suppresses inflammation and restores regenerative signaling
Oleanolic acid	Botanical antioxidant / regenerative	TNF- α , β -catenin, VEGF	Reduces inflammatory and oxidative burden while promoting regeneration
Policosanol	Bioactive anti-apoptotic / regenerative	Caspase-3, VEGF	Reduces apoptosis and enhances growth-supportive signaling
<i>Rosa rugosa</i> extract	Botanical regenerative therapy	VEGF, β -catenin	Enhances angiogenesis and Wnt-related follicular activation
FGF10-loaded nanoparticles	Targeted follicular delivery	TNF- α , IL-6, regenerative growth pathways	Suppress inflammatory cytokines and promote regrowth through localized delivery
Collagen peptide microneedles	Biomaterial-based regenerative therapy	MDA, VEGF	Reduce oxidative stress and enhance angiogenesis
Amygdalin	Bioactive anti-inflammatory therapy	TNF- α , IL-6	Attenuates inflammatory cytokine elevation and immune-mediated follicle damage

AA = alopecia areata, IL-6 = interleukin-6, JAK/STAT = Janus kinase / signal transducer and activator of transcription MDA = malondialdehyde, TNF- α = tumor necrosis factor-alpha, VEGF = vascular endothelial growth factor; Wnt = Wingless/integrated signaling pathway, β -catenin = beta-catenin.

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