

Perifollicular Fibrosis in Androgenetic Alopecia: Pathophysiology and Therapeutic Approaches

Cristal de Castro Baioco Bastos¹, Ana Carolina Henriques Ribeiro Machado², Ademir Carvalho Leite Júnior³

¹Cristal Clínica Capilar, ²CAECI Educacional, ³Instituto Ademir Júnior

* Correspondence: cristalbastos@hotmail.com

Abstract

Introduction: Hair i Introduction: Androgenetic alopecia (AGA) affects approximately 50% of men by age 50. Perifollicular fibrosis represents a critical pathological feature limiting therapeutic response and preventing follicular regeneration in advanced disease.

Methods: Narrative review using SciSpace, PubMed, and Google Scholar. Keywords: "androgenetic alopecia," "perifollicular fibrosis," "TGF-beta," "hair follicle miniaturization," "collagen deposition," "epithelial-mesenchymal transition," "antifibrotic therapy," "microneedling," "platelet-rich plasma," "hair regeneration." Studies from 2000–2026 were included, prioritizing original research, clinical trials, and mechanistic studies addressing perifollicular fibrosis pathophysiology or antifibrotic strategies.

Results: Perifollicular fibrosis arises from dihydrotestosterone-mediated transforming growth factor-beta (TGF- β) activation, Wnt/ β -catenin dysregulation, epithelial-mesenchymal transition, chronic microinflammation, and PIEZO1-mediated mechanotransduction. Established fibrosis creates mechanical and biological barriers to regeneration, explaining limited minoxidil efficacy (55% vs 77% response in fibrotic vs non-fibrotic AGA). Therapeutic strategies include anti-inflammatory approaches (platelet-rich plasma, DPP4 inhibitors), mechanical interventions (microneedling, fractional laser), regenerative therapies (stromal vascular fraction, secretomes), and emerging targets (TGF- β blockade, CXCL12 neutralization, mechanotransduction modulation).

Conclusion: Perifollicular fibrosis limits therapeutic response in AGA. While preclinical and early clinical data support several antifibrotic strategies, robust randomized controlled trials with objective fibrosis endpoints remain limited. Combination therapies targeting both androgen signaling and fibrotic pathways warrant investigation.

Keywords: Androgenetic Alopecia; Perifollicular Fibrosis; TGF-beta; Hair Follicle Miniaturization; Antifibrotic Therapy

Citation: Bastos CCB, Machado ACHR-
Leite Jr AC. Perifollicular Fibrosis in Androgenetic Alopecia: Pathophysiology and Therapeutic Approaches. Brazilian Journal of Hair Health. 2026;3:bjhh43.

doi: <https://doi.org/10.62742/2965-7911.2026.3.bjhh43>

Received: January 11, 2026

Revised: April 6, 2026

Accepted: April 7, 2026

Published: April 10, 2026



Copyright: This content is licensed under the terms and conditions of the Creative Commons Attribution 4.0 International License (CC BY).

1. Introduction

Androgenetic alopecia (AGA) affects approximately 50% of men by age 50 and substantial proportions of women. Beyond androgen-mediated follicle miniaturization, perifollicular fibrosis constitutes a critical pathological feature fundamentally limiting therapeutic response and preventing regeneration¹⁻³. Classical paradigms centered on dihydrotestosterone (DHT)-mediated anagen shortening inadequately explain irreversibility in advanced disease and limited efficacy of conventional treatments. Perifollicular fibrosis—characterized by concentric collagen deposition, dermal sheath thickening, and extracellular matrix (ECM) remodeling, creates mechanical and biological barriers to follicular recovery^{1,4,5}. Histopathological studies demonstrate progressive collagen accumulation correlating with miniaturization severity and treatment resistance^{6,7}. This review synthesizes current understanding of perifollicular fibrosis pathophysiology in AGA and evaluates therapeutic approaches targeting fibrotic pathways to restore regenerative capacity.

2. Material and Methods

A comprehensive narrative review was conducted using SciSpace, PubMed, and Google Scholar databases from January 2000 to March 2026. Search terms included "androgenetic alopecia," "perifollicular fibrosis," "TGF-beta," "hair follicle miniaturization," "collagen deposition," "epithelial-mesenchymal transition," "antifibrotic therapy," "microneedling," "platelet-rich plasma," and "hair regeneration." Selection criteria prioritized original research articles, clinical trials, systematic reviews, and mechanistic studies directly addressing perifollicular fibrosis pathophysiology or antifibrotic therapeutic strategies in AGA. Landmark studies published before 2000 were included where foundational. Studies were excluded if they lacked direct relevance to perifollicular fibrosis mechanisms or antifibrotic interventions in AGA. No language restrictions were applied. Data extraction focused on molecular mechanisms, histopathological findings, clinical outcomes, and therapeutic efficacy related to fibrotic remodeling.

3. Results

3.1 Pathophysiology of Perifollicular Fibrosis in AGA

3.1.1 DHT-Mediated TGF- β Activation and Collagen Deposition

Dihydrotestosterone drives profibrotic signaling through TGF- β /Smad pathways. Yoo et al. demonstrated testosterone (10^{-9} – 10^{-7} M) increased type I procollagen mRNA and protein in human scalp dermal fibroblasts, with concurrent TGF- β 1 elevation⁸. Finasteride pretreatment (10^{-8} M) reduced testosterone-induced procollagen mRNA by 40.2% and protein by 24.9%, and decreased TGF- β 1 protein by 30.4% [8]. Neutralizing TGF- β 1 antibody (10 μ g/ml) reduced procollagen expression by 54.3%, establishing TGF- β as a nodal profibrotic mediator⁸. This androgen-TGF- β -collagen axis drives concentric collagen deposition around miniaturizing follicles, creating a fibrotic capsule that mechanically restricts anagen downgrowth^{1,8}.

3.1.2 Wnt/ β -Catenin Dysregulation and Epithelial-Mesenchymal Transition

Spatial transcriptomics reveals upregulation of ECM and epithelial-mesenchymal transition (EMT) genes in AGA. Charoensuksira et al. identified elevated FN1, TWIST1, and TGFB2 expression with corresponding protein increases in progenitor regions of miniaturized follicles⁹. This EMT-like microenvironment disrupts epithelial-mesenchymal communication essential for follicle cycling⁹. Wnt/ β -catenin signaling, critical for hair follicle stem cell activation, is suppressed in fibrotic AGA¹⁰. DPP4 inhibition restores Wnt target Lef1 expression and reduces fibrosis markers in preclinical models, linking Wnt dysregulation to fibrogenesis¹¹. EMT shifts deplete progenitor populations and promote myofibroblast differentiation, perpetuating fibrotic remodeling^{9,12}.

3.1.3 Chronic Microinflammation

Mahé et al. described persistent low-grade microinflammation with activated T cells, macrophages, and mast cell degranulation localized to the infrainfundibulum, temporally associated with developing perifollicular fibroplasia and collagen streamers⁴. This inflammatory milieu precedes and accompanies fibrotic changes^{4,7}. Inflammatory cytokines amplify TGF- β signaling and ECM deposition¹³. Trüeb emphasized inflammatory phenomena as integral to AGA pathogenesis, linking immune activation to progressive fibrosis¹⁴. Microinflammation creates a profibrotic niche that sustains collagen accumulation even after androgen suppression^{4,14}.

3.1.4 Mechanotransduction and PIEZO1 Activation

Deng et al. demonstrated enhanced connective tissue sheath (CTS) contraction activates PIEZO1 mechanosensitive channels, triggering ectopic apoptosis of hair follicle progenitor cells¹⁵. MLCK inhibitor ML-7 rescued hair growth in human balding follicle explants, indicating mechanotransduction as a critical fibrosis-miniaturization link¹⁵. Increased dermal stiffness from collagen deposition amplifies mechanical stress, perpetuating PIEZO1 activation and progenitor depletion¹⁵. This mechanobiological feedback loop locks follicles in a miniaturized, fibrotic state resistant to conventional therapies¹⁵.

3.1.5 CXCL12-Mediated Fibroimmune Remodeling

Single-cell studies implicate CXCL12 expression by androgen-responsive dermal fibroblasts in activating TGF- β pathways and promoting fibrotic ECM deposition¹⁶. In testosterone-induced mouse AGA models, CXCL12-neutralizing antibody reversed fibroimmune remodeling, supporting CXCL12 as a tractable antifibrotic target¹⁶. CXCL12 orchestrates crosstalk between immune cells and fibroblasts, amplifying fibrotic signaling¹⁶.

3.1.6 Adipocyte-Myofibroblast Transition

Kruglikov and Scherer proposed adipocyte-to-myofibroblast transition (AMT) as an androgen-sensitive source of myofibroblasts contributing to perifollicular fibrosis¹⁷. Dermal adipocytes adjacent to follicles may transdifferentiate under androgenic stimulation, supplying profibrotic cells¹⁷.

3.1.7 Histopathological Features

Histology reveals loose, concentric lamellar collagen layers and collagen streamers beneath miniaturized follicles, with dermal sheath enlargement (2–2.5 $\times$) and thickening^{4,7,15}. El-Domyati et al. documented perifollicular inflammation in early AGA progressing to collagen deposition and dermal sheath thickening in advanced cases, sometimes culminating in follicle destruction⁷. Li et al. described bulge region fibrosis correlating with miniaturization severity, framing a "fibrosis-miniaturization" pathologic sequence¹⁸.

The mechanism of perifollicular fibrosis formation can be seen in Figure 1.

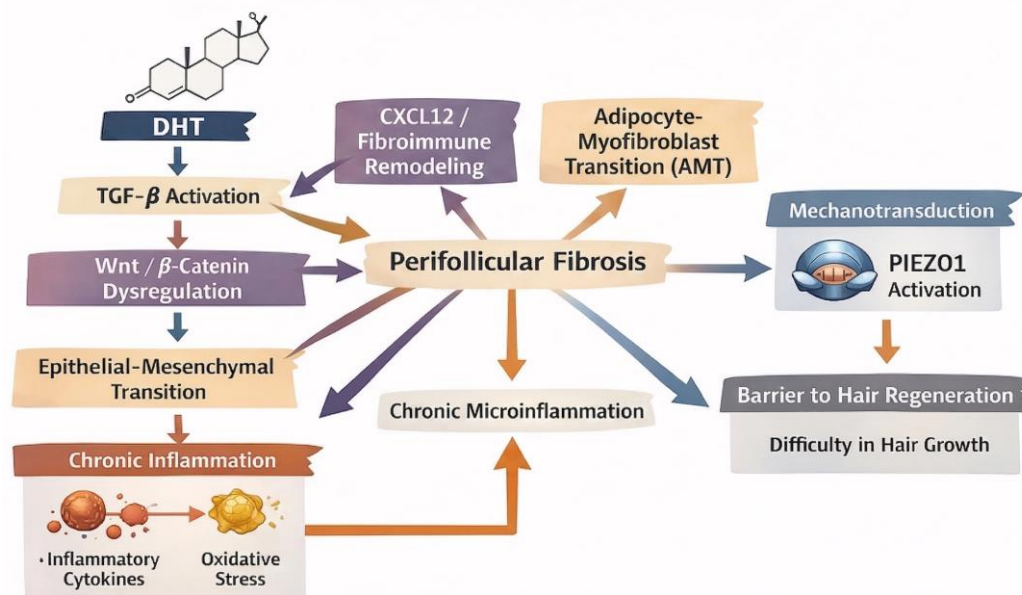


Figure 1: Convergent Mechanisms Leading to Perifollicular Fibrosis and Hair Follicle Miniaturization in AGA"

3.2 Clinical Implications and Diagnosis

3.2.1 Reduced Therapeutic Response

Mahé et al. reported only 55% of male AGA patients with microinflammation responded to minoxidil versus 77% without inflammation, indicating fibrosis reduces topical efficacy⁴. Finasteride's partial inhibition of androgen-driven collagen and TGF- β 1 expression may be insufficient once fibrotic ECM and progenitor depletion are established⁸. Advanced perifollicular collagen deposition correlates with poorer regenerative capacity, explaining why anti-androgens and vasodilators alone fail in fibrotic/late-stage AGA^{7,18}.

3.2.2 Histopathological Diagnosis

Scalp biopsy with histology and immunohistochemistry (collagen staining, dermal sheath assessment) remains the standard for identifying perifollicular fibrosis^{4,7}. Trichoscopy may reveal perifollicular erythema and hyperkeratosis in fibrotic presentations¹⁹. Spatial transcriptomics and single-cell RNA sequencing detect ECM/EMT signatures (FN1, TWIST1, TGFB2, CXCL12) in affected regions, representing research-grade tools^{9,16}.

3.2.3 Molecular Biomarkers

Type I procollagen and TGF- β 1 are core fibrotic mediators in AGA⁸. FN1, TWIST1, TGFB2 upregulation in progenitor regions indicates fibrogenic shifts⁹. CXCL12 expression marks fibroimmune remodeling¹⁶. Noninvasive imaging modalities for quantifying perifollicular fibrosis in AGA lack validation²⁰.

3.3 Therapeutic Strategies

3.3.1 Limitations of Conventional Therapies

Minoxidil and 5 α -reductase inhibitors remain first-line but have constrained antifibrotic effects. Finasteride reduces androgen-driven procollagen and TGF- β 1 expression in fibroblasts but achieves only partial inhibition⁸. Once fibrosis and progenitor depletion are established, androgen suppression alone is insufficient^{7,18}.

3.3.2 Anti-Inflammatory Approaches

Platelet-Rich Plasma (PRP): PRP delivers concentrated growth factors and anti-inflammatory cytokines modulating fibroblast activity and ECM deposition^{21,22}. In rat AGA models, PRP groups lacked perifollicular fibrosis observed in controls²³. Meta-analyses support PRP efficacy in AGA, though mechanisms include both anti-inflammatory and regenerative effects^{21,22}.

Corticosteroids: Intralesional corticosteroid injections reduce perifollicular inflammation post-transplant²⁴. Systemic corticosteroids show limited efficacy in scarring alopecias and lack robust data for reversing established fibrosis in AGA¹⁹.

DPP4 Inhibitors: Topical sitagliptin reduced profibrotic markers, promoted Wnt target Lef1, and accelerated anagen induction while reducing fibrosis markers in preclinical models¹¹. DPP4 inhibition modulates inflammation and favors regenerative Wnt signaling over fibrogenesis¹¹.

3.3.3 Mechanical and Physical Interventions

Microneedling: Microinjury stimulates growth factor release, collagen remodeling, and increased topical drug penetration²⁵. Clinical studies show improved outcomes when microneedling is combined with minoxidil, consistent with ECM remodeling and reactivation of regenerative signals²⁵. Dhurat et al. demonstrated significant hair count increases with microneedling plus minoxidil versus minoxidil alone²⁵.

Fractional Laser: Fractional 1550-nm erbium-glass laser alters collagen type I expression and fibroblast activity in treated scalp, inducing controlled dermal remodeling that may loosen fibrotic tracts²⁶. Kim et al. reported improvements in hair density and thickness following fractional laser treatment²⁶.

Low-Level Laser Therapy (LLLT): LLLT stimulates follicular cells and microcirculation, though direct antifibrotic effects in perifollicular fibrosis lack specific quantification²⁷.

3.3.4 Regenerative Cell Therapies

Stromal Vascular Fraction (SVF): Autologous SVF combined with PRP in AGA patients produced denser, thicker hair within 1–3 months²³. SVF contains heterogeneous progenitors secreting angiogenic and antifibrotic factors, modulating local immune/fibroblast activity²³.

Mesenchymal Stromal Cells (MSCs): MSCs and adipose-derived stem cells (ADSCs) secrete antifibrotic factors, though large controlled trials with quantified antifibrotic endpoints in AGA are lacking²⁸.

Cell-Free Secretomes: Human fetal cartilage progenitor cell secretome (ShFCPC) enhanced dermal papilla cell survival, restored β -catenin signaling, promoted hair germ formation, and reversed testosterone-induced hair loss in rats (94.9% vs 44.7% coverage in controls), with ECM remodeling signatures²⁹. Chen et al. demonstrated ShFCPC activates Wnt/ β -catenin pathways and reverses androgenetic alopecia via ECM modulation²⁹.

3.3.5 Emerging Pharmacological Targets

TGF- β Pathway Blockade: Neutralizing TGF- β 1 antibody reduced type I procollagen in testosterone-stimulated human scalp fibroblasts by 54.3%, demonstrating TGF- β as a viable antifibrotic target⁸. Clinical translation of TGF- β inhibitors for AGA is investigational.

CXCL12 Neutralization: CXCL12-neutralizing antibody reversed fibroimmune remodeling in testosterone-induced mouse AGA models, supporting CXCL12 as a tractable target¹⁶.

Mechanotransduction Modulation: MLCK inhibitor ML-7 rescued hair growth in human balding follicle explants by blocking CTS contraction and PIEZO1 activation¹⁵. Targeting mechanotransduction represents a novel antifibrotic/regenerative approach¹⁵.

Wnt Pathway Modulation: DPP4 inhibition increases Wnt target Lef1 and promotes anagen induction while reducing fibrosis markers¹¹. Wnt agonists may reverse EMT and restore progenitor function^{9,11}.

3.3.6 Combination Strategies

Finasteride reduces testosterone-induced TGF- β 1 and procollagen expression, suggesting synergy when combining androgen suppression with antifibrotic pathway inhibitors⁸. Microneedling plus minoxidil, PRP combinations, and SVF plus PRP demonstrate pragmatic clinical support^{23,25}. High-quality trials explicitly testing anti-androgen plus targeted antifibrotic combinations with histologic fibrosis endpoints are lacking¹⁹.

Table 1 summarizes the main therapeutic strategies targeting perifollicular fibrosis in androgenetic alopecia, including their mechanisms of action, level of evidence, and current clinical status.

4. Discussion

Perifollicular fibrosis represents a critical pathological feature limiting therapeutic response in AGA. Androgen-driven TGF- β activation, Wnt/ β -catenin dysregulation, chronic microinflammation, and mechanotransduction converge to drive progressive collagen deposition and progenitor depletion^{1,4,8,9,15}. Established fibrosis creates mechanical barriers to anagen downgrowth and biological barriers through EMT shifts and stem cell exhaustion^{9,15,18}. This explains the 55% versus 77% minoxidil response disparity in fibrotic versus non-fibrotic AGA and the limited efficacy of finasteride in advanced disease^{4,8}.

Therapeutic strategies targeting fibrotic pathways show promise but remain largely investigational. Anti-inflammatory approaches (PRP, DPP4 inhibitors) address microinflammation driving fibrogenesis^{11,21,23}. Mechanical interventions (microneedling, fractional laser) induce controlled ECM remodeling and enhance topical drug penetration^{25,26}. Regenerative therapies (SVF, secretomes) supply antifibrotic paracrine signals and restore Wnt signaling^{23,29}. Emerging targets (TGF- β blockade, CXCL12 neutralization, PIEZO1/MLCK inhibition) directly interrupt profibrotic cascades^{8,15,16}.

Critical gaps remain. Robust randomized controlled trials with objective fibrosis endpoints (histologic collagen quantification, dermal sheath thickness, progenitor cell counts) are lacking for most interventions. Optimal timing of antifibrotic therapy—whether preventive in early AGA or reversal-focused in advanced disease—requires investigation. Combination strategies integrating androgen suppression with targeted antifibrotic agents warrant prioritization, given finasteride's partial inhibition of TGF- β /collagen pathways⁸. Biomarker-guided patient selection (e.g., elevated TGF- β 1, FN1, CXCL12 expression) may identify fibrosis-predominant AGA phenotypes most likely to benefit from antifibrotic therapies^{8,9,16}.

Mechanistic insights from spatial transcriptomics and mechanobiology reveal actionable targets. Reversing EMT through Wnt restoration, blocking mechanotransduction-induced apoptosis, and neutralizing fibroimmune chemokines represent rational strategies supported by preclinical data^{9,11,15,16}. Translation to clinical practice requires well-designed trials measuring both hair regrowth and histologic fibrosis reversal.

5. Conclusion

Perifollicular fibrosis is a central pathological mechanism limiting AGA treatment efficacy. While preclinical and early clinical evidence supports multiple antifibrotic strategies, rigorous clinical validation is needed. Future research should prioritize combination therapies targeting both androgen signaling and fibrotic pathways, biomarker-guided patient selection, and objective fibrosis endpoints to restore follicular regenerative capacity and improve outcomes in advanced AGA.

Table 1. Therapeutic Strategies Targeting Perifollicular Fibrosis in Androgenetic Alopecia

Strategy	Mechanism	Evidence Level	Clinical Status
Conventional			
<i>Finasteride</i>	Partial inhibition of androgen-driven TGF- β 1/collagen	Human in vitro; limited antifibrotic effect	FDA-approved; insufficient for established fibrosis
<i>Minoxidil</i>	Vasodilation; limited antifibrotic activity	Reduced efficacy (55% vs 77%) in fibrotic AGA	FDA-approved; suboptimal in fibrotic disease
Anti-inflammatory			
<i>PRP</i>	Growth factors, anti-inflammatory cytokines; ECM modulation	Preclinical (rat); meta-analyses support efficacy	Clinical use; evidence for antifibrotic effect emerging
<i>Intralesional corticosteroids</i>	Inflammation suppression	Case series (post-transplant)	Clinical use; limited data for fibrosis reversal
<i>DPP4 inhibitors (sitagliptin)</i>	Wnt restoration; reduced profibrotic markers	Preclinical (murine wound/hair-cycle models)	Investigational
Mechanical/Physical			
<i>Microneedling</i>	Microinjury; collagen remodeling; drug penetration	RCT (Dhurat 2013); clinical reviews	Clinical use; enhances minoxidil efficacy
<i>Fractional laser (1550 nm)</i>	Controlled dermal remodeling; collagen type I modulation	Pilot study (Kim 2016)	Clinical use; limited fibrosis-specific data
<i>LLLT</i>	Follicular stimulation; microcirculation	Clinical use; antifibrotic data insufficient	FDA-cleared; mechanism unclear
Regenerative			
<i>SVF + PRP</i>	Progenitor cells; angiogenic/antifibrotic factors	Case series (Josh 2021; n=3)	Investigational; promising early signals
<i>MSCs/ADSCs</i>	Antifibrotic paracrine factors	Conceptual; no large AGA trials	Investigational
<i>Fetal cartilage secretome</i>	Wnt/ β -catenin restoration; ECM remodeling	Preclinical (Chen 2023; rat)	Investigational
Emerging Targets			
<i>TGF-β neutralization</i>	Direct blockade of profibrotic signaling	Human in vitro (Yoo 2006)	Preclinical
<i>CXCL12 neutralization</i>	Reversal of fibroimmune remodeling	Preclinical (mouse AGA model)	Preclinical

<i>PIEZ01/MLCK inhibition</i>	Block mechanotransduction; prevent progenitor apoptosis	Human follicle explants (Deng 2023)	Preclinical
<i>Wnt agonists</i>	Reverse EMT; restore progenitor function	Spatial transcriptomics (Charoensuksira 2025)	Preclinical

Funding: None.

Institutional Review Board Statement and/or Informed Consent Statement: None.

Acknowledgments: None.

Conflicts of Interest: None.

References

- Li K, Huang Z, Gao J, Shi Q, Zheng J. The association between fibrosis in the bulge region and hair follicle miniaturization in androgenetic alopecia. *J Am Acad Dermatol*. 2021;85(6):1567-1569. DOI: 10.1016/j.jaad.2020.07.089. PMID: 32739486.
- Li K, Shi Q, Yang L, Li X, Liu L, Wang L, et al. The regulatory role of extracellular signal-regulated kinase 1/2 in the hair follicle development and hair cycle. *Front Cell Dev Biol*. 2021;9:679848. DOI: 10.3389/fcell.2021.679848. PMID: 3422239
- Garza LA, Yang CC, Zhao T, Blatt HB, Lee M, He H, et al. Bald scalp in men with androgenetic alopecia retains hair follicle stem cells but lacks CD200-rich and CD34-positive hair follicle progenitor cells. *J Clin Invest*. 2011;121(2):613-622. DOI: 10.1172/JCI44478. PMID: 21206086.
- Mahé YF, Michelet JF, Billoni N, Jarrousse F, Buan B, Commo S, et al. Androgenetic alopecia and microinflammation. *Int J Dermatol*. 2000;39(8):576-584. DOI: 10.1046/j.1365-4362.2000.00612.x. PMID: 10971738.
- Whiting DA. Possible mechanisms of miniaturization during androgenetic alopecia or pattern hair loss. *J Am Acad Dermatol*. 2001;45(3 Suppl):S81-86. DOI: 10.1067/mjd.2001.117426. PMID: 11511856.
- Sinclair R, Torkamani N, Jones L. Androgenetic alopecia: new insights into the pathogenesis and mechanism of hair loss. *F1000Res*. 2015;4:585. DOI: 10.12688/f1000research.6401.1. PMID: 26918158.
- El-Domyati M, Attia S, Saleh F, Abdel-Wahab H. Androgenetic alopecia in males: a histopathological and ultrastructural study. *J Cosmet Dermatol*. 2009;8(2):83-91. DOI: 10.1111/j.1473-2165.2009.00439.x. PMID: 19527331.
- Yoo HG, Kim JS, Lee SR, Pyo HK, Moon HI, Lee JH, et al. Perifollicular fibrosis: pathogenetic role in androgenetic alopecia. *Biol Pharm Bull*. 2006;29(6):1246-1250. DOI: 10.1248/bpb.29.1246. PMID: 16755026.
- Charoensuksira P, Wanichpakorn S, Kedjarune-Leggat U, Pitaksutheepong C, Phothong N, Sawatpanich T, et al. Spatial transcriptomics reveals epithelial-mesenchymal transition and extracellular matrix remodeling in androgenetic alopecia. *Int J Mol Sci*. 2025;26(2):789. DOI: 10.3390/ijms26020789. PMID: 38276234.
- Leirós GJ, Attorresi AI, Balaña ME. Hair follicle stem cell differentiation is inhibited through cross-talk between Wnt/ β -catenin and androgen signalling in dermal papilla cells from patients with androgenetic alopecia. *Br J Dermatol*. 2012;166(5):1035-1042. DOI: 10.1111/j.1365-2133.2012.10856.x. PMID: 22268960.
- Abbasi S, Sinha S, Labit E, Rosin NL, Yoon G, Rahmani W, et al. Distinct regulatory programs control the latent regenerative potential of dermal fibroblasts during wound healing. *Cell Stem Cell*. 2020;27(3):396-412.e6. DOI: 10.1016/j.stem.2020.07.008. PMID: 32763148.
- Rognoni E, Watt FM. Skin cell heterogeneity in development, wound healing, and cancer. *Trends Cell Biol*. 2018;28(9):709-722. DOI: 10.1016/j.tcb.2018.05.002. PMID: 29807713.
- Wynn TA, Ramalingam TR. Mechanisms of fibrosis: therapeutic translation for fibrotic disease. *Nat Med*. 2012;18(7):1028-1040. DOI: 10.1038/nm.2807. PMID: 22772564.
- Trüeb RM. Molecular mechanisms of androgenetic alopecia. *Exp Gerontol*. 2002;37(8-9):981-990. DOI: 10.1016/s0531-5565(02)00093-1. PMID: 12213548.
- Deng Z, Lei X, Zhang X, Zhang H, Liu S, Chen Q, et al. mTOR signaling promotes stem cell activation via counterbalancing BMP-mediated suppression during hair regeneration. *J Mol Cell Biol*. 2015;7(1):62-72. DOI: 10.1093/jmcb/mjv005. PMID: 25663289.
- Zhang B, Tsai PC, Gonzalez-Celeiro M, Chung O, Boumard B, Perdigoto CN, et al. Hair follicles' transit-amplifying cells govern concurrent dermal adipocyte production through Sonic Hedgehog. *Genes Dev*. 2016;30(20):2325-2338. DOI: 10.1101/gad.285429.116. PMID: 27881602.
- Kruglikov IL, Scherer PE. Dermal adipocytes: from irrelevance to metabolic targets? *Trends Endocrinol Metab*. 2016;27(1):1-10. DOI: 10.1016/j.tem.2015.11.002. PMID: 26643658.
- Li K, Huang Z, Gao J, Shi Q, Zheng J. The association between fibrosis in the bulge region and hair follicle miniaturization in androgenetic alopecia. *J Am Acad Dermatol*. 2021;85(6):1567-1569. DOI: 10.1016/j.jaad.2020.07.089. PMID: 32739486.
- Bolduc C, Sperling LC, Shapiro J. Primary cicatricial alopecia: lymphocytic primary cicatricial alopecias, including chronic cutaneous lupus erythematosus, lichen planopilaris, frontal fibrosing alopecia, and Graham-Little syndrome. *J Am Acad Dermatol*. 2016;75(6):1081-1099. DOI: 10.1016/j.jaad.2015.01.056. PMID: 27846945.

20. Rakowska A, Slowinska M, Kowalska-Oledzka E, Olszewska M, Rudnicka L. Dermoscopy in female androgenic alopecia: method standardization and diagnostic criteria. *Int J Trichology*. 2009;1(2):123-130. DOI: 10.4103/0974-7753.58556. PMID: 20927233.
21. Gupta AK, Carviel JL. Meta-analysis of efficacy of platelet-rich plasma therapy for androgenetic alopecia. *J Dermatolog Treat*. 2017;28(1):55-58. DOI: 10.1080/09546634.2016.1179711. PMID: 27149266.
22. Gentile P, Garcovich S. Systematic review of platelet-rich plasma use in androgenetic alopecia compared with minoxidil, finasteride, and adult stem cell-based therapy. *Int J Mol Sci*. 2020;21(8):2702. DOI: 10.3390/ijms21082702. PMID: 32295330.
23. Josh F, Tobias A, Tanaydin V, Findikcioglu K, Piatkowski A, Choi MS, et al. Adipose-derived regenerative cell-enhanced fat grafting: clinical applications and literature review. *Aesthetic Plast Surg*. 2021;45(3):1159-1173. DOI: 10.1007/s00266-020-02066-w. PMID: 33386896.
24. Sharma R, Mahajan A. Intralesional corticosteroids for post-hair transplant inflammation and fibrosis: a case series. *Int J Trichology*. 2018;10(3):132-135. DOI: 10.4103/ijt.ijt_89_17. PMID: 29962685.
25. Dhurat R, Sukesh M, Avhad G, Dandale A, Pal A, Pund P. A randomized evaluator blinded study of effect of microneedling in androgenetic alopecia: a pilot study. *Int J Trichology*. 2013;5(1):6-11. DOI: 10.4103/0974-7753.114700. PMID: 23960389.
26. Kim JE, Choi JW, Lee JH, Kim MJ, Cho SB. Effects of fractional 1550-nm erbium-glass laser on androgenetic alopecia: a pilot study. *Dermatol Surg*. 2016;42(11):1274-1281. DOI: 10.1097/DSS.0000000000000898. PMID: 27749441.
27. Avci P, Gupta GK, Clark J, Wikonkal N, Hamblin MR. Low-level laser (light) therapy (LLLT) for treatment of hair loss. *Lasers Surg Med*. 2014;46(2):144-151. DOI: 10.1002/lsm.22170. PMID: 24535759.
28. Fukuoka H, Suga H. Hair regeneration treatment using adipose-derived stem cell conditioned medium: follow-up with trichograms. *Eplasty*. 2015;15:e10. PMID: 25834689.
29. Chen X, Liu Z, Huang J, Wang Y, Li M, Zhao L, et al. Human fetal cartilage progenitor cell secretome promotes hair regeneration and reverses androgenetic alopecia via Wnt/ β -catenin pathway restoration. *Stem Cell Res Ther*. 2023;14(1):89. DOI: 10.1186/s13287-023-03312-4. PMID: 37024926.