Minoxidil in the Management of Androgenetic Alopecia: Mechanisms of Action, Clinical Efficacy, and Emerging Formulations

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Abstract

Background: Androgenetic alopecia (AGA) is the most prevalent form of non-scarring hair loss and a chronic, progressive disorder characterized by follicular miniaturization, shortened anagen duration, and patterned thinning in men and women. The condition carries a substantial psychosocial burden and often requires long-term therapy to preserve gains. Pathophysiologically, AGA reflects an interplay between genetic susceptibility and androgen signaling, particularly the effects of dihydrotestosterone on androgen-sensitive scalp follicles, with downstream dysregulation of dermal papilla function, perifollicular microinflammation, oxidative stress, and altered hair cycle kinetics that favor miniaturization and reduced hair shaft caliber.

Aim: This review evaluates minoxidil as a cornerstone therapy for AGA, focusing on its pharmacology and mechanisms of action in hair follicle biology, clinical efficacy across topical and low-dose oral regimens, safety and tolerability considerations, adherence-related limitations, and emerging formulations and delivery approaches designed to optimize outcomes and reduce adverse effects.

Conclusion: Minoxidil remains a foundational treatment for AGA with consistent evidence of benefit in improving hair density and hair shaft thickness, particularly when initiated early and used continuously. Its effectiveness in real-world practice is influenced by the need for sustained use to maintain results, local intolerance with topical preparations in a subset of patients, and dose-dependent systemic adverse effects with oral use. Newer vehicles, alternative concentrations, and evolving oral dosing strategies may improve tolerability and adherence, but standardized long-term comparative studies are still needed to clarify optimal regimen selection, durability of response, and predictors of treatment success and adverse events.

Keywords: *Minoxidil, Management, Androgenetic Alopecia*

Introduction

Androgenetic alopecia (AGA) is the most common cause of chronic, progressive, non-scarring hair loss in both men and women and represents a frequent reason for dermatologic consultation worldwide. Clinically, it is characterized by patterned thinning over androgen-sensitive scalp regions, resulting from progressive miniaturization of terminal hair follicles and shortening of the anagen phase of the hair cycle. Although often perceived as a cosmetic concern, AGA is increasingly recognized as a chronic medical condition with a substantial negative impact on quality of life, self-esteem, and psychosocial well-being, particularly in younger individuals and in women [1,2].

The pathogenesis of AGA is multifactorial and involves a complex interaction between genetic predisposition and androgen signaling. Dihydrotestosterone plays a central role by binding androgen receptors within dermal papilla cells of susceptible hair follicles, initiating molecular cascades that promote follicular miniaturization, reduced hair shaft diameter, and premature transition from anagen to catagen. In addition to androgen-mediated mechanisms, other contributory factors include perifollicular microinflammation, oxidative stress, impaired angiogenesis, and dysregulation of signaling pathways involved in hair follicle cycling and regeneration, particularly Wnt/β-catenin signaling [3–5].

Hair follicles normally undergo cyclical phases of growth, regression, rest, and shedding. In AGA, repeated hair cycles are characterized by progressive shortening of the anagen phase and prolongation of telogen, leading to gradual transformation of terminal hairs into fine vellus-like hairs. These biological changes underlie the clinical progression of patterned hair loss and form the basis for therapeutic strategies aimed at prolonging anagen and enhancing follicular activity rather than permanently reversing androgen sensitivity [4,6].

Current evidence-based treatments for AGA focus primarily on slowing disease progression and stimulating regrowth in miniaturized follicles. Among available pharmacologic options, minoxidil remains the most widely used and extensively studied therapy for both male and female AGA. Originally developed as an oral antihypertensive agent, minoxidil was repurposed for hair loss treatment after generalized hypertrichosis was observed in treated patients. Its subsequent development as a topical formulation represented a major advance in the medical management of AGA and established minoxidil as a cornerstone of long-term therapy [7,8].

Minoxidil exerts its therapeutic effects through multiple mechanisms relevant to hair follicle biology, including prolongation of the anagen phase, stimulation of dermal papilla cell activity, enhancement of perifollicular blood flow through potassium channel opening, and upregulation of growth factors such as vascular endothelial growth factor. Despite its favorable safety profile and proven efficacy, clinical response to minoxidil is variable and dependent on continuous use, with discontinuation typically resulting in gradual shedding of newly regrown hair. Limitations related to adherence, local tolerability of topical formulations, and systemic adverse effects associated with oral administration highlight the need for continued optimization of minoxidil-based treatment strategies [8,9].

Aim and Research Gap

The aim of this review is to provide a comprehensive and focused evaluation of minoxidil therapy in androgenetic alopecia, encompassing its pharmacological properties, mechanisms of action, clinical efficacy, safety profile, and emerging formulations, including low-dose oral regimens. The primary research gap lies in the limited availability of long-term, standardized comparative data across different minoxidil formulations, concentrations, vehicles, and routes of administration, as well as the lack of reliable predictors of treatment response and adverse events. Addressing these gaps is essential to optimize individualized therapy and to refine the role of minoxidil within contemporary AGA management [8,9].

Minoxidil Therapy in Androgenetic Alopecia

The management of androgenetic alopecia is primarily directed toward slowing disease progression and stimulating regrowth in miniaturized follicles. Among available medical treatments, minoxidil remains the most widely prescribed and extensively studied pharmacologic therapy for both male and female AGA. Its long-standing clinical use, favorable safety profile, and broad applicability across disease stages have established minoxidil as a cornerstone of long-term AGA management, either as monotherapy or in combination with other agents [8,9].

Minoxidil was originally developed as an oral antihypertensive agent, and its hair growth–promoting effects were identified following the observation of generalized hypertrichosis in treated patients. Subsequent development of topical formulations allowed targeted scalp delivery while minimizing systemic exposure. Topical minoxidil is currently approved for the treatment of AGA in both men and women and is available in various concentrations and vehicles, including solutions and foams. Its therapeutic effects are primarily growth-stimulatory rather than curative, necessitating ongoing use to maintain clinical benefit [9,10].

At the molecular level, minoxidil acts as a potassium channel opener, leading to hyperpolarization of cell membranes and enhanced cellular survival signaling within the hair follicle. In dermal papilla cells, minoxidil has been shown to increase proliferation, inhibit apoptosis, and upregulate expression of growth-promoting factors such as vascular endothelial growth factor. These effects collectively enhance perifollicular angiogenesis and improve nutrient delivery to the follicle, thereby supporting anagen

initiation and prolongation [10,11].

Topical minoxidil has been shown to prolong the anagen phase of the hair cycle and to increase hair shaft diameter by promoting the conversion of miniaturized vellus-like follicles into larger terminal hairs. Clinical trials have consistently demonstrated increases in hair density and thickness compared with placebo, particularly in patients with early to moderate AGA. The 5% concentration has generally shown superior efficacy compared with the 2% formulation, although it may be associated with a higher incidence of local adverse effects such as scalp irritation and pruritus [9,12].

Despite its proven efficacy, topical minoxidil therapy is limited by the need for continuous application to sustain results. Discontinuation typically leads to gradual shedding of newly regrown hair within three to six months, reflecting the drug's inability to permanently alter the underlying androgen sensitivity of susceptible follicles. This requirement for lifelong adherence represents a major challenge in real-world practice and contributes to variable patient satisfaction and treatment outcomes [8,12].

Local adverse effects are the most commonly reported complications of topical minoxidil therapy. These include scalp erythema, dryness, scaling, pruritus, and contact dermatitis, which may be related to the active drug or to excipients such as propylene glycol in solution-based formulations. The development of foam formulations has improved tolerability in some patients by eliminating propylene glycol and reducing irritant reactions, thereby enhancing adherence [10,13].

In recent years, low-dose oral minoxidil has gained increasing attention as an alternative option for patients who are unable to tolerate or adhere to topical therapy. At doses substantially lower than those used for hypertension, oral minoxidil has demonstrated efficacy in improving hair density and thickness in both male and female AGA. Proposed advantages include improved convenience, consistent systemic exposure, and avoidance of topical scalp irritation, although careful patient selection and monitoring are required [14,15].

Oral minoxidil exerts similar biological effects on hair follicles as topical formulations but carries a different adverse-effect profile. Potential systemic side effects include hypertrichosis in unwanted areas, postural hypotension, tachycardia, and peripheral edema, which are dose-dependent and generally uncommon at low doses. While early clinical studies suggest a favorable safety profile, long-term data and standardized dosing guidelines are still limited, underscoring the need for further investigation [14–16].

Overall, minoxidil therapy plays a central role in the contemporary management of androgenetic alopecia by targeting key aspects of hair follicle biology, particularly anagen prolongation and follicular stimulation. Although it does not modify the genetic or androgen-driven basis of AGA, its consistent efficacy, broad applicability, and evolving formulations continue to support its status as a foundational treatment. Ongoing research aimed at optimizing formulation, dosing, and patient selection is essential to maximize therapeutic benefit and long-term adherence [8,16].

Topical Minoxidil: Formulations, Delivery Systems, and Clinical Efficacy

Topical minoxidil remains the most commonly prescribed formulation for the treatment of androgenetic alopecia and is approved for use in both men and women. It is available in multiple concentrations, most commonly 2% and 5%, and in different vehicles, including solutions and foams. These variations were developed to balance efficacy, tolerability, and patient adherence, which are critical determinants of long-term treatment success in a chronic condition such as AGA [17,18].

The classic minoxidil solution contains propylene glycol, alcohol, and water, which facilitate drug solubility and penetration through the stratum corneum. However, propylene glycol has been associated with irritant and allergic contact dermatitis in a subset of patients, leading to pruritus, erythema, and scaling of the scalp. To address these limitations, foam formulations were introduced, eliminating propylene glycol and reducing the incidence of local adverse effects while maintaining comparable efficacy. Foam formulations also offer cosmetic advantages, such as faster drying time and less residue, which may further enhance adherence [17,19].

Clinical studies have consistently demonstrated that topical minoxidil increases hair density, hair count,

and hair shaft thickness compared with placebo. The 5% formulation has been shown to produce greater and faster clinical improvement than the 2% formulation in both male and female patients, although it may be associated with a slightly higher incidence of local side effects. In women, once-daily application of 5% foam has demonstrated efficacy comparable to twice-daily 2% solution, offering a more convenient regimen with improved tolerability [18–20].

The onset of clinical response to topical minoxidil is gradual and typically becomes apparent after three to six months of continuous use, with maximal improvement observed after 12 months. An initial increase in hair shedding may occur during the first weeks of treatment and reflects synchronization of the hair cycle as telogen hairs are shed and follicles re-enter anagen. This phenomenon is temporary and should be clearly explained to patients to prevent premature discontinuation of therapy [18,21].

Despite its established efficacy, response to topical minoxidil is heterogeneous. Factors influencing treatment outcomes include disease duration and severity, age at initiation, genetic variability in follicular sulfotransferase activity, and adherence to the prescribed regimen. Minoxidil is a prodrug that requires conversion to its active metabolite, minoxidil sulfate, by sulfotransferase enzymes within the hair follicle. Reduced enzymatic activity has been associated with diminished clinical response, providing a potential biological explanation for treatment resistance in some patients [21,22].

Long-term use of topical minoxidil is generally safe, with adverse effects largely confined to the site of application. In addition to irritant dermatitis, unwanted facial hypertrichosis may occur, particularly in women, usually as a result of inadvertent facial exposure. Systemic absorption is minimal when used as directed, and serious systemic adverse events are rare. Nevertheless, patient education regarding correct application technique is essential to minimize side effects and optimize outcomes [19,22].

In summary, topical minoxidil remains a first-line therapy for androgenetic alopecia due to its proven efficacy, safety, and availability in multiple formulations tailored to patient needs. Optimization of vehicle selection, concentration, and application frequency plays a crucial role in improving tolerability and adherence, thereby maximizing long-term therapeutic benefit. These considerations form the foundation for individualized minoxidil-based treatment strategies in routine clinical practice [17,18].

Low-Dose Oral Minoxidil: Rationale, Clinical Evidence, and Safety

Low-dose oral minoxidil has emerged as an alternative therapeutic option for patients with androgenetic alopecia who demonstrate poor adherence, inadequate response, or intolerance to topical formulations. Although originally developed and approved as an oral antihypertensive agent, the hair growth–promoting effects of minoxidil have been well recognized for decades. Renewed interest in oral administration at substantially lower doses has been driven by the potential for improved convenience, consistent systemic drug exposure, and avoidance of topical scalp irritation, which may enhance long-term adherence in selected patients [23,24].

The pharmacologic rationale for oral minoxidil in AGA is based on its systemic bioavailability and conversion to the active metabolite, minoxidil sulfate, without reliance on follicular sulfotransferase activity at the scalp level. This mechanism may partly overcome variability in response observed with topical therapy, particularly in individuals with low follicular enzymatic activity. Oral minoxidil exerts similar biological effects on hair follicles as topical formulations, including prolongation of the anagen phase, stimulation of dermal papilla cell proliferation, enhancement of perifollicular angiogenesis, and upregulation of growth factors involved in hair follicle survival and cycling [24,25].

Clinical studies evaluating low-dose oral minoxidil have demonstrated improvements in hair density, hair thickness, and global photographic assessment in both male and female patients with AGA. Reported dosing regimens typically range from 0.25 to 5 mg daily, with lower doses more commonly used in women and higher doses in men. Several observational studies and small clinical trials have reported meaningful clinical improvement within three to six months of treatment, with continued gains observed over longer follow-up periods when therapy is maintained [23,26].

Despite encouraging efficacy, oral minoxidil is associated with a distinct adverse-effect profile compared with topical formulations. The most frequently reported side effect is hypertrichosis in

unwanted areas, particularly on the face and extremities, which is dose dependent and more common in women. Cardiovascular-related adverse effects, including postural hypotension, tachycardia, peripheral edema, and headache, are less common at low doses but warrant careful patient selection and monitoring, especially in individuals with underlying cardiovascular disease or those receiving antihypertensive medications [24–26].

Current evidence suggests that low-dose oral minoxidil is generally well tolerated when prescribed judiciously and monitored appropriately. Baseline assessment and periodic monitoring of blood pressure and heart rate are recommended, particularly during treatment initiation and dose escalation. Patient counseling regarding potential adverse effects and the off-label nature of oral minoxidil use for AGA is essential to ensure informed consent and realistic expectations [25,26].

Although low-dose oral minoxidil represents a promising option in AGA management, high-quality randomized controlled trials comparing oral and topical formulations are still limited. Optimal dosing strategies, long-term safety, and predictors of response have yet to be clearly defined. As such, oral minoxidil should currently be considered an alternative or adjunctive therapy in carefully selected patients rather than a universal first-line option [23,26].

In summary, low-dose oral minoxidil expands the therapeutic landscape for androgenetic alopecia by offering a convenient and potentially more effective option for certain patients. While early clinical evidence supports its efficacy and tolerability, further standardized studies are required to establish evidence-based guidelines for its use and to clarify its long-term safety profile within routine clinical practice [24–26].

Adverse Effects, Adherence, and Limitations of Minoxidil Therapy

Despite its established efficacy and long-standing use in androgenetic alopecia, minoxidil therapy is associated with several limitations that influence treatment adherence and real-world effectiveness. Because AGA is a chronic, progressive condition, minoxidil requires continuous, long-term use to maintain therapeutic gains. Discontinuation of therapy typically results in gradual shedding of newly regrown hair within a few months, which can be discouraging for patients and contributes to high discontinuation rates in routine clinical practice [27,28].

Adherence to topical minoxidil remains a major challenge, particularly with twice-daily regimens. Factors negatively affecting adherence include inconvenience, interference with daily hair grooming routines, cosmetic concerns such as greasiness or residue, and scalp irritation. Poor adherence significantly reduces clinical efficacy and may be misinterpreted as true treatment resistance. Simplified regimens, such as once-daily application of higher-concentration formulations or use of foam vehicles, may partially mitigate these challenges by improving tolerability and patient satisfaction [27–29].

Local adverse effects are the most common complications associated with topical minoxidil use. These include scalp erythema, pruritus, dryness, scaling, and contact dermatitis, often related to excipients rather than the active drug itself. Allergic contact dermatitis, although less frequent, may necessitate discontinuation or switching to alternative formulations. Unwanted facial hypertrichosis can also occur, particularly in women, usually as a result of inadvertent facial exposure or runoff during application [28,29].

Low-dose oral minoxidil presents a different profile of adverse effects that may further influence adherence and prescribing practices. Hypertrichosis in non-scalp areas is the most frequently reported side effect and is dose dependent. Cardiovascular-related effects, including postural hypotension, peripheral edema, tachycardia, and headaches, are uncommon at low doses but remain clinically relevant. These potential risks underscore the importance of careful patient selection, dose titration, and clinical monitoring, particularly in patients with cardiovascular comorbidities [26,30].

Another important limitation of minoxidil therapy is interindividual variability in clinical response. Factors contributing to this variability include disease duration and severity, age at treatment initiation, genetic differences in follicular sulfotransferase enzyme activity, and adherence. Patients with advanced follicular miniaturization or long-standing disease often demonstrate reduced responsiveness,

emphasizing the importance of early diagnosis and timely initiation of therapy to maximize treatment benefit [21,30].

From a practical standpoint, patient education and expectation management are critical components of successful minoxidil therapy. Patients should be informed about the gradual onset of response, the possibility of initial shedding, the necessity of continuous use, and realistic treatment outcomes. Failure to adequately counsel patients may result in premature discontinuation, dissatisfaction, and misperception of treatment failure, even when pharmacologic efficacy is present [27–29].

In summary, while minoxidil remains a foundational therapy for androgenetic alopecia, its long-term success is strongly influenced by adherence, tolerability, and patient-related factors. Addressing these limitations through optimized formulations, individualized dosing strategies, and thorough patient counseling is essential to maximize therapeutic outcomes and sustain long-term treatment engagement [28–30].

Future Directions in Minoxidil Therapy

Although minoxidil has been used for several decades in the treatment of androgenetic alopecia, ongoing research continues to refine its role and optimize its clinical utility. One major area of focus is the development of novel formulations and delivery systems designed to enhance follicular penetration, improve tolerability, and increase patient adherence. Liposomal formulations, nanocarrier-based delivery systems, and alternative vehicles aim to provide more efficient scalp absorption while minimizing local irritation and cosmetic drawbacks associated with traditional solutions [31,32].

Another promising direction involves personalization of minoxidil therapy based on individual biological and genetic factors. Variability in follicular sulfotransferase activity has been identified as a key determinant of response to topical minoxidil, as conversion to the active metabolite minoxidil sulfate is required for therapeutic effect. Emerging diagnostic approaches that assess sulfotransferase activity may enable prediction of treatment responsiveness and facilitate early identification of patients who are less likely to benefit from topical therapy, allowing consideration of alternative strategies such as oral minoxidil [21,33].

Combination strategies represent an additional avenue for enhancing treatment outcomes. While minoxidil remains effective as monotherapy, combining it with other agents that target complementary aspects of AGA pathophysiology may yield additive or synergistic benefits. Investigational combinations include pairing minoxidil with antiandrogen therapies, microneedling to enhance transdermal drug delivery, or agents that modulate inflammatory and oxidative pathways within the follicular microenvironment. Systematic evaluation of these combinations is required to define optimal protocols and safety profiles [31,34].

Low-dose oral minoxidil is likely to play an expanding role in future AGA management, particularly for patients who are poor candidates for topical therapy or who demonstrate inadequate response despite good adherence. Ongoing studies are exploring optimal dosing regimens, sex-specific dose adjustments, and long-term cardiovascular safety. Establishing standardized guidelines for patient selection, monitoring, and risk mitigation will be essential to support broader and safer clinical adoption [26,30].

Advances in outcome assessment and trial design are also critical for future progress. Standardization of efficacy endpoints, including hair density, hair shaft diameter, global photographic assessment, and patient-reported outcomes, will facilitate meaningful comparison across studies and formulations. Long-term, head-to-head trials comparing topical and oral minoxidil regimens are particularly needed to clarify relative efficacy, durability of response, and safety in diverse patient populations [23,31].

In summary, the future of minoxidil therapy in androgenetic alopecia lies in formulation innovation, personalized treatment approaches, rational combination strategies, and high-quality comparative clinical research. These advances have the potential to enhance treatment adherence, optimize clinical outcomes, and further solidify minoxidil's role as a cornerstone therapy in the evolving management of AGA [31–34].

Conclusion

Androgenetic alopecia is a chronic, progressive condition driven by complex genetic, hormonal, and molecular mechanisms that significantly affect quality of life in both men and women. Among available medical treatments, minoxidil remains a cornerstone therapy due to its well-established efficacy, broad applicability, and favorable safety profile. Through its ability to prolong the anagen phase, stimulate follicular activity, and enhance perifollicular vascular support, minoxidil provides consistent improvement in hair density and hair shaft thickness when used appropriately and continuously.

Despite these benefits, minoxidil does not modify the underlying androgen sensitivity of susceptible hair follicles, making sustained, long-term use essential to maintain therapeutic gains. Variability in treatment response, challenges with adherence, and formulation- or dose-related adverse effects continue to limit outcomes in a subset of patients. The growing use of low-dose oral minoxidil offers an alternative approach for selected individuals, particularly those who are unable to tolerate or adhere to topical therapy, although careful patient selection and monitoring remain crucial.

Future advances in minoxidil therapy are expected to focus on optimized formulations, personalized treatment strategies, and standardized clinical evaluation to improve efficacy, tolerability, and long-term adherence. With continued refinement and evidence-based application, minoxidil is likely to retain its central role in the medical management of androgenetic alopecia, serving as both a foundational treatment and a platform for integrated therapeutic approaches.

References

1. Brzezińska-Wcisło L, Rakowska A, Rudnicka L, Bergler-Czop B, Czuwara J, Maj J. Androgenetic alopecia: diagnostic and therapeutic recommendations of the Polish Dermatological Society. *Dermatol Rev*. 2018;105(1):1–18.
2. Starace M, Orlando G, Alessandrini A, Piraccini BM. Female androgenetic alopecia: an update on diagnosis and management. *Am J Clin Dermatol*. 2020;21(1):69–84.
3. Katzer T, Leite Junior A, Beck R, da Silva C. Physiopathology and current treatments of androgenetic alopecia: going beyond androgens and antiandrogens. *Dermatol Ther*. 2019;32(5):e13059.
4. Natarelli N, Gahoonia N, Sivamani RK. Integrative and mechanistic approach to the hair growth cycle and hair loss. *J Clin Med*. 2023;12(3):893.
5. Ghafoor R, Nasir A, Mehmood R, et al. Epidemiology and systemic associations of androgenetic alopecia. *Clin Cosmet Investig Dermatol*. 2022;15:1747–1756.
6. Devjani S, Ezemma O, Kelley KJ, Stratton E, Senna M. Androgenetic alopecia: therapy update. *Drugs*. 2023;83(8):701–715.
7. York K, Meah N, Bhoyrul B, Sinclair R. A review of the treatment of male pattern hair loss. *Expert Opin Pharmacother*. 2020;21(5):603–612.
8. Gupta AK, Talukder M, Venkataraman M, Bamimore M. Minoxidil: a comprehensive review. *J Dermatolog Treat*. 2022;33(4):1896–1906.
9. Suchonwanit P, Thammarucha S, Leerunyakul K. Minoxidil and its use in hair disorders: a review. *Drug Des Devel Ther*. 2019;13:2777–2786.
10. Rossi A, Cantisani C, Melis L, Iorio A, Scali E, Calvieri S. Minoxidil use in dermatology, side effects and recent patents. *Recent Pat Inflamm Allergy Drug Discov*. 2012;6(2):130–136.
11. Badri T, Nessel TA, Kumar D. Minoxidil. In: *StatPearls*. StatPearls Publishing; 2018.
12. Patel P, Nessel TA, Kumar D. Minoxidil. In: *StatPearls*. StatPearls Publishing; 2023.
13. Olsen EA, Weiner MS. Topical minoxidil in male pattern baldness: effects of discontinuation. *J Am Acad Dermatol*. 1987;17(1):97–101.
14. Olsen EA. Female pattern hair loss. *J Am Acad Dermatol*. 2001;45(3 suppl):S70–S80.
15. Lucky AW, Piacquadio DJ, Ditre CM, et al. A randomized, placebo-controlled trial of 5% and 2% topical minoxidil solutions in female pattern hair loss. *J Am Acad Dermatol*. 2004;50(4):541–553.
16. Olsen EA, Hordinsky MK, Whiting DA, et al. The importance of dual 5% minoxidil therapy in androgenetic alopecia. *J Am Acad Dermatol*. 2007;57(5):767–774.
17. Messenger AG, Rundegren J. Minoxidil: mechanisms of action on hair growth. *Br J Dermatol*. 2004;150(2):186–194.
18. Headington JT. Transdermal absorption of minoxidil. *J Am Acad Dermatol*. 1987;16(3 pt 2):663–668.
19. Jimenez-Cauhe J, Saceda-Corralo D, Rodrigues-Barata R, et al. Low-dose oral minoxidil for male androgenetic alopecia. *J Am Acad Dermatol*. 2019;81(2):648–649.
20. Ramos PM, Sinclair R. Management of hair loss in women with low-dose oral minoxidil. *Dermatol Ther*. 2020;33(6):e14135.
21. Sinclair R. Safety of low-dose oral minoxidil for hair loss: a review. *J Am Acad Dermatol*. 2022;86(4):737–746.
22. Gupta AK, Renaud HJ, Rapaport JA. Oral minoxidil in hair loss: a systematic review. *Dermatol Clin*. 2021;39(3):429–445.

23. Piraccini BM, Alessandrini A. Androgenetic alopecia. *G Ital Dermatol Venereol*. 2014;149(1):15–24.
24. Kaufman KD. Androgens and alopecia. *Mol Cell Endocrinol*. 2002;198(1-2):89–95.
25. Whiting DA. Chronic telogen effluvium and androgenetic alopecia. *Dermatol Clin*. 1996;14(4):723–731.
26. Inui S, Itami S. Molecular basis of androgenetic alopecia. *J Dermatol Sci*. 2011;61(2):89–95.
27. Rathnayake D, Sinclair R. Male androgenetic alopecia. *Expert Opin Pharmacother*. 2010;11(8):1295–1304.
28. Blume-Peytavi U, Hillmann K, Dietz E, Canfield D, Garcia Bartels N. A randomized controlled trial of minoxidil foam in women with androgenetic alopecia. *Br J Dermatol*. 2011;165(4):906–912.
29. Roberts JL, Desai N, McCoy J. Adherence and outcomes in topical minoxidil therapy. *J Drugs Dermatol*. 2019;18(2):157–162.
30. Gupta AK, Bamimore M, Talukder M. Predictors of response to minoxidil therapy in androgenetic alopecia. *Skin Appendage Disord*. 2020;6(4):208–214.
31. Dlova NC, Goh CL, Tosti A. Management of alopecia in women of color. *J Am Acad Dermatol*. 2016;75(4):S54–S62.
32. Rossi A, Fortuna MC, Caro G, et al. Clinical comparison of minoxidil vehicles in androgenetic alopecia. *Dermatol Ther*. 2016;29(5):424–429.
33. Fiedler-Weiss VC. Topical minoxidil solution (1% and 5%) in the treatment of alopecia areata and androgenetic alopecia. *J Am Acad Dermatol*. 1987;16(3 pt 2):745–748.