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Androgenetic alopecia: an international expert view on the aetiopathogenesis, quality of life and current and emerging therapeutic approaches

Androgenetic alopecia (AGA) or pattern hair loss is a non-scarring hair condition defined by gradually reduced, miniaturized hair follicles. It is a multifactorial disorder primarily triggered by dysregulation of the hair cycle due to alterations between the crosstalk of numerous cell subpopulations. *Objective*: To provide a focus on existing and novel management options, preceded by a short overview of the current knowledge about AGA. *Material and Methods*: A group of international experts in AGA analysed 85 literature sources about AGA (retrieved from PubMed and Google Scholar and published between 2000 and 2025) and discuss current and novel treatment options. *Results*: Recent research evidenced that targeting the hair follicle and the surrounding tissue, rather than solely relying on solutions offered by minoxidil, finasteride, or other products, provides a safe and highly beneficial improvement of AGA. The authors underscore the significant scientific value and crucial role of advanced dermocosmetics with proven clinical efficacy in the long-term management of AGA, in addition to pharmacological active ingredients or other treatment options. These innovations offer compelling early intervention strategies, potentially delaying reliance on lifelong pharmaceutical regimens and optimizing the overall patient journey and quality of life. *Conclusion*: In addition to current treatment options of AGA, novel topical non-pharmacological therapeutic options are available, helping to safely and efficiently treat this multifactorial hair condition.

Keywords: Androgenetic alopecia, aetiology, physiopathology, quality of life, treatment, therapeutic approach

Androgenetic alopecia (AGA) is a non-scarring, genetic and androgen-dependent hair disease defined by gradually reduced miniaturized hair follicles. It is a multifactorial disorder primarily triggered by dysregulation of the hair cycle due to alterations between the crosstalk of numerous cell subpopulations [1].

Other factors, including chronic micro-inflammation and oxidative stress characterise AGA [2].

AGA is one of the most common chronic hair problems seen by dermatologists worldwide [3]. About 30% to 50% of men and around 30% of middle-aged women suffer from AGA [4, 5]. It is the most frequent type of hair loss in men [6, 7]. Due to its highly visible appearance, patients with AGA may suffer from anxiety and depression [8, 9].

In 2017, the European Dermatology Forum released evidence-based guidelines to support dermatologists in choosing safe and effective treatments for AGA [10]. These guidelines propose other primary and complementary management options to treat AGA. However, they are currently outdated as, since their enforcement, new management options have become available.

We herein provide a short overview of the aetiopathogenesis of AGA, impact on QoL and burden as well as of existing management options. Moreover, we focus on a novel management approach for AGA.

Material and methods

Four experts in hair conditions met during the European Hair Research Society Congress in Warsaw in May 2025 to discuss AGA, namely from the point of view of current and upcoming treatment options. The authors performed a non-systematic analysis of the literature on AGA and identified 85 literature sources about AGA from PubMed and Google Scholar published between 2000 and 2025. Key search words included: androgenetic alopecia and pattern hair loss alone or combined with

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one or multiple of the following: hormones, physiopathology, quality of life, burden, treatment.

Results

Aetiology

AGA has a polygenic inheritance. It involves a genetic predisposition, an increased level of androgens as well as other non-androgenic factors.

Increased follicle sensitivity to androgens is an important causative factor, which explains the therapeutic effectiveness of antiandrogens [11, 12]. In addition to intrinsic factors (genetic, ageing, hormones), development or worsening of AGA may also be due to extrinsic factors such as diet, stress, smoking and other environmental factors [13, 14].

Physiopathology

AGA is linked to androgens and genetic predisposition with an abnormal sensitivity of scalp hair follicles to circulating androgens due to an increased activity of androgen receptors [15, 16]. Androgens alter mesenchyme-epithelial cell interactions within the follicle. As a result, hair growth, dermal papilla size, dermal papilla cells, keratinocyte and melanocyte activities are impacted [17, 18]. Despite scientific advances, the causal molecular mechanisms of androgen-related actions remain to be elucidated.

The activation of androgen receptor gradually shortens the anagen phase leading to a reduced size of the hair follicle (follicular miniaturization). Hair follicles become thinner and shorter and produce thinner and shorter hair shafts [19, 20]. Another hypothesis considers that the miniaturization of the follicle may be due to the reduction in the number of hair follicle stem cells in the hair bulge or dermal sheath [21].

This reduction may be caused by the loss of contact between the arrector pili muscle and the bulge, leading to a disruption of the stem cell function [22]. Likewise, microinflammation plays an important role in the pathogenesis of AGA. Inflammatory cells infiltrate the follicular bulb and cause progressive fibrosis of the perifollicular zone, leading to injury of follicular stem cells, impairment of the normal hair cycle, and finally cause hair follicle miniaturization [16].

Protein HIF-1 acts as an oxygen sensor to adapt the cell behavior to the environment and promote cell regeneration when necessary. It is expressed in all hair follicles and is highly involved in their cyclic regeneration. HIF-1 is continuously exposed to prolyl hydroxylase and asparaginyl-hydroxylase, causing its degradation. In AGA and hair miniaturization, this pathway is altered with a faster miniaturizing of the hair follicle compared to healthy ones, failing to stimulate cell regeneration and hair growth.

Outside the hair follicle, premature hair loss was shown to be associated with a thickening of the sheath around the hair follicle [23]. This thickening is considered to be related to the accumulation and the rigidification of collagen that restrains the hair root from anchoring itself deeply in the dermis [24]. The hardened sheath compresses the follicle and inhibits microcirculation that provides nutrients to the hair. Consequently, the miniaturized follicle produces a thinner, weakened fibre resulting in premature hair loss.

Clinical and histological diagnosis

In women, AGA (or female pattern hair loss) results in a decreased hair density and thickness in the frontal area, with a sparse pattern and preservation of the frontal hairline [25-30]. In men, AGA (or male pattern hair loss) involves the temporal areas causing regression of the temporal hair line, the anterior area, and the vertex [4]. Bi-temporal recession has been reported in 64% of women, especially after menopause, compared to 99% in men. Conversely, mid-frontal alopecia affects almost 2/3 of women after the age of 80 years, while 3/4 of Caucasian men over the 80's present with mid-frontal and vertex hair loss [31].

Follicular miniaturization initially involves adjacent follicles to different extents: the same area contains hair of normal caliber, hair slightly thinner, and very thin and short hair. In severe forms of AGA, the miniaturization affects follicles diffusely and the hair shafts are uniformly thin [7].

The reduced number of hair also appears with the decrease of hair shafts per follicular unit. This feature is best seen when comparing trichoscopy of androgen-sensitive areas of the scalp with that of the occipital area [7]. Peripilar depressions, also called 'peripilar halos', appear as dark halos extending approximately 1 mm around the follicular ostium from which the hair emerges. They are specific and characteristic of AGA and are more often seen in mild forms. The pathogenesis of these brown perifollicular halos is not known; they are assumed to reflect the presence of inflammation of the superficial perifollicular dermis, often present in the scalp of patients with AGA [32].

Histologically, AGA is characterized by signs of follicle destruction, including follicular miniaturization, perifollicular inflammation, and fibrotic tracts, also called follicular stelae [33].

Clinical assessment of AGA involves invasive and non-invasive methods, such as scalp dermoscopy, pull tests, and scalp biopsies [34].

Trichoscopy (scalp dermoscopy) allows diagnosis of mild, early forms of AGA and patient monitoring during follow-up [35]. The dermoscopic features of AGA include variation of the hair shaft diameter, empty follicle phenomenon, reduced number of hair per follicular unit and peripilar depressions.

Quality of life (QoL) and burden

AGA patients frequently suffer from feelings of anxiousness, helplessness, and diminished self-esteem [36]. This psychosocial stress often gives rise to behavioural coping efforts. Their dissatisfaction with their hair leads to an overall body image dissatisfaction and a concomitant reduction in QoL [37].

The psychosocial impact seems to be more severe in women when compared to men. The chronicity of AGA

and its therapeutic refractoriness increase the psychosocial stress and highly impacts the women's QoL [38, 39]. Conversely, only 25% of men with AGA consider their hair loss to be extremely upsetting, with 65% stating to be only modestly to moderately distressed emotionally [40].

Management

The effective management of AGA remains challenging. An inventory of the most frequently used treatment options, as well as a focus on novel therapeutic approaches, is given hereafter.

Current treatment options

Minoxidil

Topical minoxidil remains the gold standard treatment [41].

Minoxidil is available as topical solution and foam, with the 5% solution being approved for treatment of male AGA and the 2% for female AGA [42-44]. Low-dose oral minoxidil (2.5-5 mg/d for male, and 0.25-1.25 mg/d for female AGA) is effective and safe, but it is an off-label prescription and should be used with caution in patients with cardiovascular diseases [45, 46].

The effects of minoxidil on hair growth are first visible after 6–8 weeks, and become maximal at 12–16 weeks [47]. If successful, continuous treatment is required to maintain efficacy [10].

However, the efficacy of minoxidil in AGA varies greatly. Goren *et al*. reported that after 16 weeks of continuous topical application of minoxidil 5%, less than 40% of male patients respond [48]. Among women, 13-20% of patients report a moderate effectiveness with 2% minoxidil [49]. It has been estimated that 60% of male patients may not respond to topical minoxidil therapy because of decreased baseline levels of the sulfotransferase enzyme required to activate minoxidil to its active metabolite [48].

Finasteride

Finasteride is an inhibitor of type 2 5 alpha-reductase (SRD5A2). It has been approved by the FDA to treat adult men with mild to moderate AGA at an oral dosage of 1 mg/d. It decreases serum, prostate, and scalp dihydrotestosterone (DHT) by 60-70% [50]. It inhibits the gene *SRD5A2* by forming a stable complex with the enzyme in the presence of Nicotinamide Adenine Dinucleotide Phosphate (NADPH). SRD5A2 inhibitors prevent the hydride transfer from NADPH to testosterone, responsible for the conversion of testosterone to DHT. Effectiveness was evidenced after 6 months, and the efficacy peak was reached after 12-15 months. Drug withdrawal was followed by gradual loss of all effects.

Topical 0.25% finasteride solution applied once daily similarly decreased scalp DHT levels and induced hair regrowth in AGA [51, 52].

Some studies suggest that higher doses of oral finasteride (2.5 to 5 mg/d) are necessary for female AGA, but caution is required due to the risk of teratogenicity in premenopausal women [53, 54]. Side effects may result from interactions with proteins beyond SRD5A2 inhibition [55].

Off-label treatments and procedures

Dutasteride, low-level laser therapy, platelet-rich plasma and microneedling are procedures that are used off-label to treat AGA. They are all expensive when compared to other options and act more as boosters for stimulating the hair follicle activity than for a chronic reversal of AGA. Hair follicle autologous transplantation is an effective but costly surgical technique that requires skilled surgeons [56].

Dutasteride

Dutasteride, a synthetic 4-azasteroid, is a selective and competitive inhibitor of both type 1 and type 2 isoenzymes of 5α-reductase. Oral dutasteride is effective in AGA [57-61]. It is not approved for AGA in Europe and the USA. However, its off-label use at 0.5 mg/d or every other day is considered by international guidelines as a second-line treatment, recommended for male patients when previous treatment with 1 mg finasteride for over 12 months has been ineffective [10].

Low-level laser therapy

Low-level laser therapy (LLLT) or photomodulation represents a relatively recent and efficacious AGA treatment option. LLLT uses light with wavelengths between 600 and 1100 nm to stimulate hair regrowth. LLLT is believed to foster hair growth by promoting anagen-phase re-entry of telogen hair follicles, increasing the duration of anagen phase, and preventing premature conversion of anagen hairs to catagen-phase hairs [62]. Due to its non-invasive nature, LLLT is becoming popular in AGA. However, according to current guidelines, it should only be used complementary therapy [10]. Further research is required to establish the efficacy of LLLT in AGA compared with established therapies and to evaluate its long-term use.

Platelet-rich plasma

Platelet-rich plasma (PRP) contains chemokines, growth factors, cell signalling molecules and cytokines with important implications in the control and regulation of many cellular processes. Among the components of PRP, alpha granules produced and stored in platelets are relevant. These granules contain platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), transforming growth factor beta (TGFβ), and insulin-like growth factors (IGF) [63, 64]. While results with PRP are promising, there is still a lack of standardization of protocols, which remains a barrier in adequately characterizing the therapeutic response of patients [56]. Current guidelines affirm that there is no standardized technique, and therefore PRP treatment is not recommended in AGA [10].

Microneedling

Microneedling is thought to stimulate growth factors, activate follicular bulb stem cells, and increase expression of genes associated with hair growth [4]. Moreover, it is thought to stimulate hair growth via upregulation of Wnt/ß-catenin signalling and induction of VEGF [65].

Hair follicle unit transplantation

Hair follicle unit transplantation (FUT) is a surgical procedure consisting of removing and transplanting hair follicles, mainly from units in the occipital region to those concerned by AGA. The transplanted follicles do not miniaturize, grow in groups of 1 to 4 hairs and are harvested as units. It should only be considered in patients with sufficient donor hair [10, 66].

Combination treatments

A systematic review of randomized controlled trials concluded that a combination of minoxidil 2% and systemic finasteride 1 mg is superior to each treatment alone [67].
Another systematic review of the combination of LLLT and minoxidil therapy reported mixed results in the literature concerning the efficacy of this combined therapy [68].
A meta-analysis compared minoxidil monotherapy to minoxidil plus LLLT combination therapy [67, 69]. While there was a considerable heterogeneity in the studies, the combination (LLLT and minoxidil) therapy demonstrated superior efficacy to monotherapy.

Other management options

Other treatment options, mainly for mild AGA, include the use of ketoconazole, zinc pyrithione and 2,4-diamino-pyrimidine-N-oxide [56, 70-72].
2,4-diamino-pyrimidine-N-oxide prevents collagen rigidification through the inhibition of the lysyl-hydroxylase, which is involved in collagen maturation or rigidification [73]. Due to this inhibition, there is less collagen reticulation, fibres remain loose, and there is no stiffening of the connective tissue sheath. Therefore, hair roots are less compressed, microcirculation is less inhibited, and hair roots are more firmly anchored in the dermis, preventing hair loss.
Table 1 provides a summary of the evidence level, efficacy to prevent progression and capacity of improvement for the most common therapeutic interventions. *Table 2* proposes a list of other AGA management options.

Nutritional supplements and dietary options

Nutritional supplements in AGA, especially those that are hypothesized to inhibit 5-alpha reductase, stimulate IGF-1 production, decrease oxidative stress, and provide essential nutrients or blood flow to hair follicles, have

Table 1. Summary of evidence level, efficacy to prevent progression and/or improve androgenetic alopecia, safety aspects and practicability for the most common therapeutic interventions according to the Guidelines issued by Kanti *et al*. in 2017 [10].

	Level of evidence	Efficacy to prevent progression	Efficacy to improve	Safety	Practicality (patient)	Practicality (physician)
Male patients						
Finasteride 1 mg QD	1	+++	++	+++	++++	++
Dutasteride 0.5 mg QD*	1	+++	+++	++	++++	++
Minoxidil 5% BID	1	+++	++	++++	+/++	+++
(solution, foam)	2	-	+++	++	+ intervention	+
Hair transplantation with/					+++ long-term	
without combination	2		+/	++	+++	+++
treatment	3	+/	+/	+	+/-	+
		+/				
LLLT						
PRP						
Female patients						
Minoxidil 2% BID solution	1	+++	++	++++	+	+++
Minoxidil 5% QD foam						
Hormones oral in hyperan-	3	+	+	+	+++	
drogenism	3	+/	+/-	+	+++	++
Hormones oral in normal	4	-	++	++	+ intervention	++
hormones	2				+++ long-term	+
Hair transplantation	3	+/-	+/-	++	+++	
		+/	+/	+	+/-	+++
LLLT						+
PRP						
Global consideration	Poor	⟷				Good
	-	+/-	+	++	+++	++++

This table provides a rough orientation. According to Kanti et al., an individualized therapy concept, taking into account clinical status, demands, needs and complaints of each patient, is mandatory. The physician and patient must decide together on the best-suited individualized therapy, considering available evidence, expected therapeutic results, practicality and compliance.

Table 2. Miscellaneous agents, products and interventions, grouped according to their assumed main mechanism of action [10].

Claimed mechanism of action	Active	Level of evidence	Studies included (n)	Grade of evidence	Mode of application	Part of combination product
1-DHT-inhibitory activity	β-sitosetrol	3	1	B	Oral	Yes
	Biochanin A	3	1	B	Topical	No
	Polysorbate 60	2	1	A2	Topical	No
	Serenoa repens	3	1	B, B	Oral	Yes, No
		3	1	B	Topical	Yes
	Curcuma *aeruginosa*	2	1	A2	Topical	Yes, No
	Biochanin A	3	1	B	Topical	No
2-Anti-inflammatory activity	Ketoconazole	3	1	B	Topical	Yes
	Roxithromycin	3	1	B	Topical	No
	Zinc pyrithione	2	2	B, A2	Topical	Yes, No
3-Improved perifolicular	Glycerolesters and silicium	3	B	B	Topical	Yes
		2	A2	A2	Topical	Yes
	Niacin derivates	2	A2	A2	Topical	No
	Prostaglandins (viprostol, latanoprost)					
4-Improved hair follicle nutrition	Amino acids (cysteine, histidine)	3	1	B	Topical	Yes
			1	B	Oral	Yes
		3	2	B	Oral	Yes
	Vitamins (biotin, niacin)	3	1	B	Oral	Yes
	Trace elements (zinc, copper)					
5-Not precisely reported or unknown mechanism of action	Adenosine	3	1	B	Topical	No
	Biotin	3	1	B	Oral	Yes
		2	1	A2	Topical	
	Hibiscus	3	1	B	Topical	Yes
	Marine extract and	2	1	A2	Oral	No
	silica component	4	1	C	Topical	No
	Melatonin	3	1	B	Oral	Yes
	Millet seed	2	2	A2	Topical	Yes
	Niacin derivates	3	1	B	Topical	No
	Proanthocyanidins	4	1	C	Topical	Yes
	Red ginseng	2	2	B, A2	Topical	Yes, No
	Tretinoin	2	1	A	Topical	No
	Valproic acid					
Interventions						
Not precisely reported or unknown mechanism of action	Botulinum toxin	4	1	C	Scalp injections	No
	Electromagnetic/-static field	1	4	A2, A2, B, B	Device	Yes, No

Level of Evidence: 1: Studies grade A1 evidence or studies with mainly consistent results grade A2 evidence, 2: Studies grade A2 evidence or studies with mainly consistent results grade B evidence, 3: Studies grade B evidence or studies with mainly consistent results grade C evidence, 4: Little to missing systematic evidence.

Grade of evidence: A1: Meta-analysis which includes at least one randomized clinical trial of grade A2 evidence with consistent results of the different studies, A2: Randomized, double-blind, comparative clinical studies of high-quality (e.g. sample size calculation, flow chart of patient inclusion, ITT-analysis, sufficient size), B: Randomized, clinical studies of lesser quality or other comparable studies (not-randomized, cohort- or case–control studies), C: Non-comparable studies, D: Expert opinion.

Additional reported actives without clinical studies included in the guideline comprise: aloe, Aminexil, bergamot, caffeine, ciclosporin, cimicifuga racemosa, ginkgo, mesotherapy and sophora.

been more or less successfully tested for their efficacy [74]. Moreover, antioxidants (including vitamins E and C) have shown some efficacy in treating female AGA by increasing hair density and thickness. Protein-based supplements and probiotics have been shown to be effective in treating AGA by providing essential nutrients and increasing blood flow to hair follicles [56].

New management approaches

Despite the multiple proposed management options, none really provides a satisfying outcome. Management is long-lasting, and until today, there are no real alternatives to the gold standard minoxidil, especially for non-responders [49].

Within the integrative health framework, a novel therapeutic approach targets both intra- and extra-follicular mechanisms, such as HIF-1 and hair anchorage, thus providing a more comprehensive therapeutic approach for AGA. According to the experts, the limited patient adherence to minoxidil, coupled with scarce clinical evidence in larger female populations, compromises optimal therapeutic outcomes and renders new treatment strategies necessary.

Recently, cortexolone 17α-propionate (clascoterone), a novel androgen receptor antagonist that competes with DHT binding sites on cytoplasmic androgen receptors, has been made available to treat acne [75]. It is currently undergoing phase II clinical trials for the treatment of AGA [76].

While most of the currently available management options, including minoxidil, promote hair growth by improving nutrient delivery to hair follicles, or act on a hormonal level (such as finasteride), a novel approach targets the hair follicle by limiting the deactivation of HIF-1. This allows for an increased hair growth and a decreased hair loss in both men and women, and also reduces collagen rigidification in the tissue surrounding the hair follicle (*figure 1*).

Several compounds have been reported to modulate the HIF-1 pathway, including niacinamide, [6]-gingerol extract, quercetin, *Gingko biloba* extract, desferrioxamine and an iron chelator inhibiting PHD, increasing the HIF-1 α expression, and promoting hair regeneration [77-82]. An unpublished *in vitro* study showed a synergistic effect of niacinamide and [6]-gingerol on the expression levels of the HIF-1 target genes *CA9,* and to a less extent, *BNIP3* in keratinocytes.

Biotinoyl tripeptide-1 (Procapil™, Biotinyl-GHK, Croda Inc, USA) is a derivative of GHK for penetration improvement. It signals for restructuring of the dermis, which reduces collagenous material and reactivates stem cell niches and improves follicle anchoring thickening via extracellular matrix (ECM) remodelling [83]. Moreover, a biomimetic peptide combining acetyltetrapeptide-3 and red clover flower extract rich in Biochanin A (Capixyl™, Lucas Meyers Cosmetics, USA) restructures the dermis, promotes the renewal of the ECM and the synthesis of attachment proteins [84].

A yet unpublished clinical multicentre study in 40 female and 37 male patients showed that the continuous once-daily use of a novel fixed serum combination of 4-diamino-pyrimidine-N-oxide, niacinamide 3% and [6]-gingerol extracted from ginger roots as well as biotinoyl tripeptide-1 and a biomimetic peptide (Aminexil® Clinical R.E.G.E.N Booster, Vichy Laboratoires, France) resulted, both in men and women, in a significantly (p<0.05) increased anagen hair density with 6 weeks, continuing until the 12th week. In parallel, the telogen hair percentage significantly (p<0.05) decreased within 6 weeks and continued to significantly (p<0.05) decrease until week 12. The tested serum showed a similar efficacy with improved cosmeticity profile compared to the standard AGA treatment applied twice daily. The serum was very well tolerated and accepted by the patients of the study.

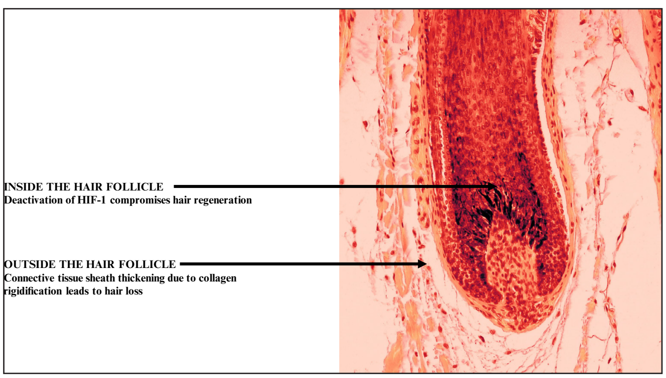

Figure 1. Proposed novel therapeutic approach in androgenetic alopecia.

HIF-1: hypoxia-inducible factor-1.
HIF-1 is deactivated by prolyl-hydroxylase and asparaginyl-hydroxylase.

Discussion

Treatment of AGA remains a major issue [85].

While several management options are currently available, none really provides satisfying results. Even minoxidil, the gold standard treatment for AGA, provides, according to the experts' opinion, only limited success due to reduced compliance to the treatment, mainly due to its proposed formulation. Moreover, its use at 5% is not recommended in women, and thus limits treatment options in this patient population. Other treatments may cause side effects, while other treatment options are currently not available, very expensive (procedures) or used off-label [52].

In addition to the available pharmacological treatments, other products, including dermocosmetics, vitamins and nutritional supplements are proposed alone or as adjunctive, but again, a significant gap exists regarding substantial clinical studies that could sufficiently corroborate their efficacy [10, 56].

In 2017, a group of experts proposed evidence-based guidelines for the treatment of AGA [10]. To date, these guidelines are outdated, and ever since novel treatment options have become available.

Recent research assessed a novel management approach in targeting the hair follicle and the surrounding tissue instead of providing solutions offered by minoxidil and finasteride or other products. Early clinical investigations showed that this novel approach increases HIF-1 expression inside the hair follicle and improves the quality of the surrounding tissue by softening the peri-follicular collagen, restructuring the dermis and reactivating stem cells. As such, it is a promising novel option for AGA that can be used alone in milder forms, or in case of failure of minoxidil treatment, or as an adjunct to pharmacological treatments, aiming to increase hair growth and reduce hair loss as a multitargeting management approach.

The authors underscored the significant scientific value and crucial role of advanced dermocosmetics with proven clinical efficacy in the long-term management of AGA. These innovations offer compelling early intervention strategies, potentially delaying the reliance on lifelong pharmaceutical regimens and optimizing the overall patient journey and quality of life.

In conclusion, in addition to current treatment options of AGA, novel topical non-pharmacological therapeutic options are becoming available, helping to safely and efficiently treat this multifactorial hair condition. ■

Disclosure

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