Adjunctive approaches for androgenetic alopecia: A clinical review of nutritional, light-based, topical, and lifestyle interventions

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Androgenetic alopecia (AGA) is a chronic, progressive condition with limited Food and Drug Administration—approved treatments. Therefore, there is increasing interest in adjunctive options among patients and physicians. This clinical review synthesizes and critically appraises current evidence on nutritional supplements and nutraceuticals, light-based therapies, topical agents, and lifestyle modifications to guide their integration into evidence-based AGA management. Mechanistic and histologic studies indicate that inflammatory, oxidative, and microvascular alterations may contribute to follicular miniaturization, supporting interest in adjunctive strategies that promote scalp homeostasis. Among reviewed interventions, some demonstrate modest benefit, whereas many others lack adequate data. Significant gaps persist, including small sample sizes, heterogeneous methodologies, limited long-term safety data, and inconsistent outcome measures. Social media and direct-to-consumer hair loss companies remain major sources of AGA misinformation, underscoring the need for high-quality digital patient education and physician-guided resource curation. Future independently funded randomized controlled trials and mechanistic studies are essential to define optimal use parameters and support evidence-based, individualized, multimodal AGA therapy for patients. (J Am Acad Dermatol 2026;95:148-61.)

Key words: adjuvants; androgenetic alopecia; hair loss; light-based therapy; nutritional supplements; topicals.

INTRODUCTION

Androgenetic alopecia (AGA) is the most common hair loss type.[1,2] Since US Food and Drug Administration (FDA)—approved treatments for AGA are limited (topical minoxidil for both sexes, oral finasteride for males), patients and physicians are increasingly interested in adjuvant approaches to augment first-line interventions.[3] However, evidence-based guidelines remain limited, with frequent misinformation on press and social media.[2,4-11] In this clinical review, we aim to critically appraise adjuvant approaches for AGA into a unified, evidence-based approach, to help physicians adapt to the rapidly evolving treatment paradigm and deliver optimal care.

MECHANISTIC FOUNDATIONS

AGA is a chronic, progressive form of hair loss characterized by follicular miniaturization and shortened anagen phase driven by dihydrotestosterone-mediated signaling in androgen-sensitive dermal papilla fibroblasts.[1,2,7,12-14] Since inflammatory, oxidative, and microvascular factors contribute to pathogenesis,[12,15-23] adjunctive approaches may complement first-line agents by reducing

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inflammation, restoring vascularity, and supporting cellular resilience within the scalp microenvironment.[1,2,7,12,15]

EVIDENCE REVIEW OF ADJUVANT APPROACHES & ASSOCIATED SIDE EFFECT PROFILES

Nutritional supplements & nutraceuticals

Essential nutrients. Biotin (vitamin B7) is an essential carboxylase cofactor involved in metabolic pathways, including mitochondrial carboxylases in hair roots.[24,25] There is limited evidence for treatment of hair loss, except in rare cases of true biotin deficiency, valproic acid–induced alopecia, and uncombable hair syndrome.[24-30] Biotin interferes with laboratory tests dependent upon biotin-streptavidin immunoassays, including troponins, thyroid function tests, and pregnancy tests.[27,29-31] Given lack of evidence and interference with laboratory tests, biotin is not supported for AGA treatment.

Notably, in a retrospective study of 138 female and male nonscarring alopecia patients (66 AGA patients), 50.7% had low iron, vitamin D, zinc, or thyroid abnormalities, but supplementation did not significantly improve hair density or caliber, suggesting limited benefit from micronutrient repletion alone.[32] Therefore, current data do not support iron, vitamin D, or zinc supplementation as AGA treatment (Table I).

Multivitamins/combination supplements. A popular hair nutraceutical brand offers sex-specific and age-specific formulations containing proprietary plant-derived ingredients, including saw palmetto extract (SPE), ashwagandha, and tocotrienols,[31,38-40] with some clinical studies showing positive results.[40-45] Additionally, Feldman et al's Bayesian network meta-analysis (NMA) of 17 female and male randomized controlled trials (RCTs) found that this nutraceutical increased terminal hair (TH) density by mean difference of 7.32 TH/cm^2 vs placebo at 24 weeks in women with AGA.[46] However, the overall certainty of evidence was limited and improvements appeared to level off after 12 weeks,[46] suggesting less consistent benefit compared with established therapies.[46]

An oral marine protein-derived supplement composed of proprietary shark and mollusk proteins, biotin, vitamin C, zinc, and procyanidin B2 reduced hair shedding and increased hair volume, thickness, and scalp coverage in clinical trials of patients with self-perceived thinning hair.[31,38,39,47-52] In Feldman et al's Bayesian NMA, this nutraceutical demonstrated modest TH regrowth in men (mean difference 13.23 TH/cm^2 vs placebo) at 24 weeks, but improvements stabilized after 12 weeks, indicating limited efficacy vs other AGA treatments.[46]

CAPSULE SUMMARY

- There are limited Food and Drug Administration–approved treatments for androgenetic alopecia, driving interest in alternative options.
- Evidence supporting adjunctive approaches for androgenetic alopecia is heterogenous, with modest benefit observed in selected interventions.
- Synthesis of evidence is needed to guide multimodal treatment and avoid misinformation.

Another hair nutraceutical contains SPE, L-cystine, biotin, taurine, zinc, horsetail extract, and B vitamins.[31,38] An RCT of 35 male AGA patients found that this nutraceutical increased the anagen/telogen ratio by 23.4% at 6 months vs baseline.[53] Another RCT of 80 female and male alopecia patients (75% AGA, 25% telogen effluvium) found that hair density increased by 11.6 hairs/cm^2 after 6 months of supplementation vs placebo ($P < .001$).[54] Despite demonstration of modest efficacy for these nutraceuticals in AGA, many studies were limited by subjective inclusion criteria of self-perceived hair thinning, no baseline nutritional status or definition of hair loss pattern, small sample sizes, and manufacturer funding. Therefore, current evidence does not support supplementation with these nutraceuticals for adjunctive AGA therapy (Table II).

Natural oils and plant-based compounds. SPE, a botanical 5-alpha reductase inhibitor (5ARI), has been marketed for hair support due to its mild antiandrogenic properties.[31,55-61] An RCT of 80 female and male AGA patients randomized to oral SPE 100 mg/day, oral placebo, topical SPE 20% lotion 5 mL/day, or topical placebo for 16 weeks found that both oral and topical SPE treatment decreased hair shedding and increased hair density vs baseline, whereas placebo increased hair shedding and decreased hair density vs baseline (all $P < .05$).[56] Only oral SPE demonstrated increased hair thickness vs baseline (19.61 μm vs 16.89 μm, $P = .017$).[56] Another RCT of 100 male AGA patients found that SPE 320 mg/day for 2 years stabilized hair loss, but was less effective than finasteride 1 mg/day (AGA improvement in 38% vs 68% of patients, no P value reported).[62] Additionally, Gupta et al's NMA of 24 male AGA studies evaluating conventional and nonconventional therapies (oral/topical 5ARIs, oral/topical minoxidil, topical botanicals, and oral marine complex supplementation) found that topical SPE demonstrated limited efficacy for hair growth (Surface Under the Cumulative Ranking curve [SUCRA] = 14.1%).[63,64]

Abbreviations used:

5ARI:	5-alpha reductase inhibitor
AGA:	androgenetic alopecia
FDA:	Food and Drug Administration
LLLT:	low-level light therapy
NAFL:	nonablative fractional laser
NMA:	network meta-analysis
PSO:	pumpkin seed oil
RCT:	randomized controlled trial
SPE:	saw palmetto extract
SUCRA:	Surface Under the Cumulative Ranking curve
TH:	terminal hair

Reported adverse events include sexual side effects (impotence in 1% of patients in benign prostatic hyperplasia study) and decreased prostate cancer markers, teratogenicity, and hematologic and gastrointestinal side effects in case reports.[31,55,65-71] Since oral vs topical SPE has demonstrated superior efficacy,[56] oral SPE is a reasonable adjunctive option, although more robust studies are warranted to clarify efficacy and optimal dosage.

Pumpkin seed oil (PSO) is another botanical 5ARI.[31,55,58,59,61,72] An RCT of 76 male AGA patients found that treatment with PSO 400 mg/day vs placebo for 24 weeks increased hair counts (40% vs 10% increase, $P < .001$) and demonstrated higher patient satisfaction (10-point scale: 3.5 vs 2.3, $P = .003$).[73] Another RCT of 60 female AGA patients treated with topical PSO 1 mL/day or topical minoxidil 5% 1 mL/day found improvement after 3 months in both groups in hair shaft diversity (both $P < .001$), vellus hair (PSO: $P < .001$, minoxidil: $P = .02$), and regrowing hair (both $P < .001$), with minoxidil more effective for hair regrowth ($P = .005$).[74] Evidence supports adjunctive PSO for AGA, especially for patients opposed to prescription 5ARIs, although prospective studies are needed to better define safety, efficacy, and adverse/teratogenic effect profile.

Rosemary oil is a botanical 5ARI with antioxidant, antibacterial, antifungal, and anti-inflammatory properties.[52,55,59,61,63,72] An RCT of 100 male AGA patients found that twice-daily topical rosemary oil lotion increased hair counts at 6 months, similar to 2% minoxidil ($P > .05$), with scalp itching less frequent in the rosemary group ($P < .05$).[75] In Gupta et al's NMA of 24 male AGA studies, topical rosemary oil 1 mL twice/day showed moderate efficacy for hair growth (SUCRA = 53.6%), but less than oral finasteride, topical finasteride, and topical minoxidil 5% and greater than oral minoxidil 5 mg, topical procyanidin 0.7%, and SPE.[63] However, SUCRA-based rankings in this NMA reflect probabilistic ordering rather than confirmed clinical superiority and should be interpreted cautiously given indirect comparisons and heterogeneity in study design.[63] In a Canadian Delphi-based consensus study evaluating 45 interventions for female and male AGA patients, rosemary oil was not recommended (82% consensus against due to insufficient evidence).[76] Data demonstrate that rosemary oil is a well-tolerated adjunct for AGA, particularly for curlier hair, as daily use can leave straight hair greasy (Table III).

Light-based therapies

Low-level light therapy. Low-level light therapy (LLLT), or photobiomodulation, uses red/near-infrared light to stimulate tissue healing, with studies showing scalp hair regrowth.[12,78-85] RCTs have demonstrated improvements in hair density, thickness, and coverage without major adverse effects in both male and female AGA patients.[86-95] An RCT of 91 male AGA patients treated with 1 mL topical minoxidil 5% twice/day or 3 10-minute LLLT sessions/week reported increased hair density and improvement in clinical photography vs baseline (both $P < .001$) and similar between groups (hair density: $P = .42$, photography: $P = .17$) at 6 months.[96] Several meta-analyses corroborated these findings, supporting efficacy of LLLT in enhancing hair growth in AGA patients, despite variability in device design and study methodology.[46,80,97-100] Consensus studies and guideline perspectives support LLLT as a safe and effective second-line therapy for AGA, although results are most reliable for devices with clinically validated parameters in RCTs.[76,79,82,101] Although there is no standardized LLLT protocol for AGA due to variability among FDA-cleared devices, studies generally report treatment efficacy for 10-minute to 30-minute sessions 3-5 times/week using red (620-670 nm) or near-infrared wavelength devices consistently for 4-6 months.[76,79,82,101] While generally considered safe, potential risks include eye injury from direct laser exposure, scalp irritation, and lack of long-term data. Based on current evidence, LLLT represents a reasonable adjunct for AGA, especially for patients reluctant to take systemic therapies or pregnant/lactating patients, as it is noninvasive and well tolerated.

Fractional and nonablative lasers. Fractional and nonablative lasers have emerged as adjunctive treatments for AGA, inducing controlled dermal microinjury that increases blood flow and activates wound-healing pathways, growth factor release, and follicular regeneration.[78,81,83,84,102] Clinical studies have shown positive results,[103-108] including a retrospective, observational study of 132 female and male alopecia patients (43.2% AGA) in which blinded reviewers correctly identified post-treatment improvement in 96.9% of

Table I. Summary of the reviewed essential nutrients

Adjunct	Relevance to AGA	Studies	Findings	Level of evidence*
Biotin	Essential cofactor for mitochondrial carboxylases in hair roots	Randomized, open-label, crossover trial of 10 healthy males treated with oral biotin alone, topical minoxidil alone, or combination biotin/minoxidil[33]	HGR stable between baseline and with supplementation with biotin alone (2.50 mm/week baseline vs 2.47 mm/week 14 days, $P = .53$), but increased with combination therapy (2.36 mm/week baseline vs 2.64 mm/week 14 days, $P = .02$).	IIB
Iron	Deficiency associated with hair loss	Retrospective study of 155 female hair loss patients[32,34,35]	70.3% of patients had ferritin < 60 ng/mL and found that patients whose ferritin levels increased by $> 40\%$ with iron supplementation were more likely to report subjective improvement in hair growth	III
Zinc	Cofactor for metabolic enzymes involved in hair growth; deficiency associated with hair loss	RCT of 73 female hair loss patients treated with combination oral zinc and calcium, zinc or calcium alone, or topical minoxidil[36]	Increased hair thickness in the zinc group ($P = .01$), greatest hair density increase from baseline in the minoxidil group ($P = .001$), and greatest patient satisfaction in the combination therapy group	IIB
Vitamin D	Regulator of gene expression involved in hair follicle cycling and growth; deficiency associated with presence and severity of AGA	Randomized comparative study of 45 female AGA patients treated with combination oral vitamin D (5000 IU/day) and topical minoxidil 5% twice/day or either therapy alone for 6 months[37]	Combination therapy improved Ludwig's grade ($P = .002$), but not vitamin D alone ($P = .05$)	IIB

AGA, Androgenetic alopecia; *HGR*, hair growth rate; *RCT*, randomized controlled trial.

*Levels of evidence are based on the *Journal of the American Academy of Dermatology* guidelines and reflect study design rather than strength of clinical efficacy: level IA evidence includes evidence from meta-analysis of randomized controlled trials; level IB evidence includes evidence from ≥ 1 randomized controlled trial; level IIA evidence includes evidence from ≥ 1 controlled study without randomization; level IIB evidence includes evidence from ≥ 1 other type of experimental study; level III evidence includes evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies; and level IV evidence includes evidence from expert committee reports or opinions, or clinical experience of respected authorities, or both.

Table II. Summary of the reviewed nutraceuticals

Adjunct & relevance to AGA	Studies	Findings	Level of evidence*
Nutraceutical containing SPE, ashwagandha, marine collagen, and other ingredients optimized to target hormonal, inflammatory, and oxidative mechanisms contributing to AGA	Several RCTs of female and male patients with self-perceived thinning hair treated with nutraceutical vs placebo, including recent RCT of 112 male patients who underwent treatment vs placebo for 6 months[40-45]	Improvements in subjects taking the supplement vs placebo in hair growth ($P < .01$) and quality ($P < .05$). Studies limited by subjective inclusion criteria of self-perceived hair thinning with no baseline nutritional status and funding by the product's manufacturer	IB
	NMA of 17 female and male RCTs[46]	Nutraceutical increased TH density by a mean difference of 7.32 TH/cm^2 vs placebo at 24 weeks in women with AGA, with low confidence (23.4%) and an attenuated response after 12 weeks	IA
Nutraceutical hair thickening supplement containing oral marine proteins, L-cysteine, biotin, vitamin C, zinc, and procyanidin B2 shown to trigger cell proliferation and signaling at the dermal papilla of the hair follicle	Several RCTs of female and male patients with self-perceived thinning hair treated with nutraceutical vs placebo[47-51]	Reductions in hair shedding and increases in hair volume, thickness, and scalp coverage. Studies limited by subjective inclusion criteria of self-perceived hair thinning, no baseline nutritional status or definition of the hair loss pattern, small sample sizes, and funding by the product's manufacturer	IB
	NMA of 17 female and male studies[46]	Nutraceutical demonstrated modest TH regrowth in men (mean difference 13.23 TH/cm^2 vs placebo) at 24 weeks, but its effect plateaued after 12 weeks and was not statistically different from placebo	IA
Nutraceutical containing SPE, L-cysteine, biotin, taurine, zinc horsetail extract, and B vitamins to support hair growth and improve hair density	RCT of 35 male AGA patients treated with nutraceutical or placebo for 6 months[53]	Nutraceutical treatment increased the anagen/telogen ratio by 23.4% at 6 months vs baseline	IIB
	RCT of 60 AGA and 20 telogen effluvium female and male patients treated with nutraceutical or placebo for 6 months[54]	Hair density increased by 11.6 hairs/cm^2 after 6 months of nutraceutical supplementation vs placebo ($P < .001$)	IIA

AGA, Androgenetic alopecia; *NMA*, network meta-analysis; *RCT*, randomized controlled trial; *SPE*, saw palmetto extract; *TH*, terminal hair.

*Levels of evidence are based on the *Journal of the American Academy of Dermatology* guidelines and reflect study design rather than strength of clinical efficacy: level IA evidence includes evidence from meta-analysis of randomized controlled trials; level IB evidence includes evidence from ≥ 1 randomized controlled trial; level IIA evidence includes evidence from ≥ 1 controlled study without randomization; level IIB evidence includes evidence from ≥ 1 other type of experimental study; level III evidence includes evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies; and level IV evidence includes evidence from expert committee reports or opinions, or clinical experience of respected authorities, or both.

evaluable cases, supporting FDA clearance of a 1565 nm nonablative fractional laser (NAFL) for adults with alopecia.[109] Additionally, an RCT of 30 male AGA patients treated with either 4 sessions of 1565 nm NAFL (every 2 weeks) or 1 mL topical minoxidil 5% twice/day for 10 weeks reported increased hair density and average TH diameter vs baseline for both treatment groups (both $P = .001$), with NAFL treatment showing greater improvements in hair counts and hair density vs minoxidil ($P < .05$).[110] However, these findings should be interpreted with caution, as topical minoxidil is not expected to demonstrate maximal clinical effect within 10 weeks. Two meta-analyses demonstrated efficacy of fractional laser treatment for female and male AGA patients, as monotherapy or adjuvant therapy.[111,112] Reported adverse effects included pain, pruritus, erythema, edema, and xerosis.

Other topical agents. Procyanidin B_2, melatonin, and caffeine have been investigated as adjunctive modalities for AGA.[31,52,63,113] Topical procyanidin 0.7%, an anti-inflammatory flavonoid derived from apple juice, applied twice/day increased total hair counts at 6 months in 43 male AGA patients vs placebo (6.6 hairs/cm^2 vs −7.2 hairs/cm^2, $P < .001$), with a total increase of 23 hairs/cm^2 vs baseline after 12 months of procyanidin treatment ($P < .005$).[114] An open-label trial of 1891 female and male AGA patients treated with topical melatonin (antioxidant neurohormone produced in hair follicles) 0.0033% nightly for 3 months reported greater proportion of patients with negative hair-pull tests vs baseline (12.2% vs 61.5%, $P < .001$).[115] An open-label trial of 210 male AGA patients found that 6 months of topical caffeine 0.2% solution 2 mL twice/day vs topical minoxidil 5% 1 mL twice/day demonstrated similar increase in anagen hairs vs baseline (+11.68% vs +10.59%, $P = .574$).[116] These topicals demonstrate potential as well-tolerated adjuncts for AGA.

Topical ketoconazole 2%, an antifungal with antiandrogenic properties, has shown benefit in male AGA by reducing scalp *Malassezia* colonization and inflammation,[117-119] but appeared less effective in Gupta et al's NMA (SUCRA = 30%) compared with topical melatonin (SUCRA = 55.4%) and procyanidin 0.7% (SUCRA = 48.6%).[63] Several RCTs have found that topical ketoconazole 2% shampoo increases hair shaft diameter and hair density in male AGA, largely within combination regimens.[119-123] Consensus studies describe ketoconazole as a well-tolerated adjuvant or third-line treatment for AGA (Table IV).[76,101]

Lifestyle & behavioral modifications

Emerging evidence suggests that behavioral factors may influence AGA disease course, offering opportunities for adjunctive lifestyle interventions.[52,124,125] Chronic stress has been associated with hair cycle dysregulation,[126] and stress-reduction strategies including psychotherapy may provide indirect benefit by modulating inflammatory and neuroendocrine pathways.[52] Additionally, prospective studies have investigated scalp massage to increase blood flow and enhance follicular signaling to promote hair regrowth with limited success.[127,128] Dietary patterns may further modulate AGA, as diets rich in antioxidants and protein, including the Mediterranean diet (fruits/vegetables, whole grains, legumes, olive oil, and fish), may promote hair growth in AGA, whereas the restrictive human chorionic gonadotropin diet (severe caloric restriction with human chorionic gonadotropin supplementation) and high-glycemic diets and insulin resistance may be associated with AGA.[124,129-133] Smoking is associated with increased risk, earlier onset, and greater severity of AGA through vascular, oxidative, and hormonal mechanisms.[134-136] Therefore, smoking cessation counseling may be relevant, although improvement after cessation has not been clearly demonstrated.[134-136] Lifestyle optimization may serve as a supportive measure in AGA management, pending more robust data.

COMBATTING MISINFORMATION

Patients frequently seek information regarding hair loss on social media and direct-to-consumer hair loss platforms, yet most alopecia-related content is created by nonmedical sources rather than board-certified dermatologists.[5,6,137-141] Furthermore, limited prioritization of large-scale regulatory or FDA-supported AGA research may reflect its classification as a cosmetic concern despite evidence demonstrating its negative impact on quality of life,[1,10,12] contributing to gaps in high-quality evidence and propagation of unverified treatment claims. Physicians could direct patients to credible resources and integrate curated digital education tools, such as high-quality, brief evidence-based videos into routine AGA management.[11]

FUTURE DIRECTIONS

Given current limitations, adjuncts are best incorporated using a patient-centered, physician-guided framework, with the most clinically meaningful approaches and their potential use scenarios summarized in Table V. These treatment options may be considered after optimization of first-line therapies, including topical/oral minoxidil, prescription 5ARIs, and spironolactone.[142-144] Future research should prioritize large, independently funded RCTs to analyze comparative efficacy, safety, and optimal use parameters of adjunctive AGA therapies in both females and males. Standardization of outcome measures and treatment protocols, including clearly defined clinical and

Table III. Summary of the reviewed natural oils and plant-based compounds

Adjunct	Relevance to AGA	Studies	Findings	Level of evidence*
Saw palmetto extract (SPE)	Botanical 5ARI	RCT of 80 female and male AGA patients treated with either oral or topical SPE or placebo for 16 weeks[56]	Oral and topical SPE decreased hair shedding and increased hair density vs baseline ($P < .05$). Oral SPE demonstrated increased hair thickness vs baseline (19.61 μm vs 16.89 μm, $P = .017$)	IIA
		RCT of 100 male AGA patients treated with SPE or finasteride for 2 years[62]	SPE stabilized hair loss, but is less effective than finasteride (38% of patients experienced improvement vs 68% with finasteride)	IB
		NMA of 24 male AGA studies[63]	Topical SPE demonstrated limited efficacy in treating male AGA (SUCRA = 14.1%)	IA
Pumpkin seed oil (PSO)	Botanical 5ARI	RCT of 76 male AGA patients treated with PSO or placebo for 24 weeks[73]	PSO treatment vs placebo resulted in increased hair counts (40% increase vs 10% increase, $P < .001$) and patient satisfaction (10-point scale: 3.5 vs 2.3, $P = .003$)	IIA
		RCT of 60 female AGA patients treated with topical PSO or topical minoxidil for 3 months[74]	Significant improvement in both groups regarding hair shaft diversity (both $P < .001$), vellus hair (PSO: $P < .001$, minoxidil: $P = .02$), and regrowing hair (both $P < .001$), although minoxidil demonstrated more significant efficacy vs PSO ($P = .004$)	IIA
Rosemary oil	Botanical 5ARI with antioxidant, antibacterial, antifungal, and anti-inflammatory properties	RCT of 100 male AGA patients treated with topical rosemary oil lotion or topical minoxidil twice/day for 6 months[75]	Significant increase in hair count at 6 months in both groups ($P > .05$), with scalp itching occurring less frequently in the rosemary group ($P < .05$)	IB
		NMA of 24 male AGA studies[63]	Topical rosemary oil showed moderate efficacy (SUCRA = 53.6%), performing below oral/topical finasteride and topical minoxidil 5% but outperforming several other agents	IA
		Canadian Delphi consensus study evaluating 45 interventions for female and male AGA[76]	Rosemary oil was not recommended by the expert panel (82% consensus against)	IV
Tea tree oil	Antiseptic and anti-inflammatory properties	Pilot study of 32 male AGA patients with a multimodal microemulsion of minoxidil, diclofenac, and tea tree oil or minoxidil alone or placebo for 32 weeks[77]	The multimodal microemulsion significantly improved hair count, thickness, and patient-reported outcomes compared with minoxidil alone or placebo	IIB
Castor oil	Prostaglandin D2 inhibition	Frequently promoted on social media and within hair care communities as a remedy for hair loss, but there are no clinical studies supporting its efficacy for treating AGA[72]		IV

5ARI, 5-alpha reductase inhibitor; *AGA*, androgenetic alopecia; *NMA*, network meta-analysis; *PSO*, pumpkin seed oil; *RCT*, randomized controlled trial; *SPE*, saw palmetto extract; *SUCRA*, Surface Under the Cumulative Ranking curve.

*Levels of evidence are based on the *Journal of the American Academy of Dermatology* guidelines and reflect study design rather than strength of clinical efficacy: level IA evidence includes evidence from meta-analysis of randomized controlled trials; level IB evidence includes evidence from ≥ 1 randomized controlled trial; level IIA evidence includes evidence from ≥ 1 controlled study without randomization; level IIB evidence includes evidence from ≥ 1 other type of experimental study; level III evidence includes evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies; and level IV evidence includes evidence from expert committee reports or opinions, or clinical experience of respected authorities, or both.

Table IV. Summary of other reviewed topical agents

Topical adjunct	Relevance to AGA	Studies	Findings	Level of evidence*
Procyanidin 0.7%	Anti-inflammatory flavonoid	Double-blind RCT of 43 male AGA patients treated with topical procyanidin twice/day vs placebo for 6 months[114]	Topical procyanidin vs placebo resulted in increased total hair counts (6.6 hairs/cm^2 vs −7.2 hairs/cm^2, $P < .001$), with a total increase of 23 hairs/cm^2 vs baseline after an additional 6 months of procyanidin treatment ($P < .005$)	IIB
		NMA of 24 male AGA studies[63]	Topical procyanidin showed moderate efficacy (SUCRA = 48.6%), performing above topical ketoconazole, but below topical melatonin and topical rosemary oil	IA
Melatonin 0.0033%	Antioxidant neurohormone produced in hair follicles	Open-label trial of 1891 female and male AGA patients treated with topical melatonin nightly for 3 months[115]	Greater proportion of patients with negative hair-pull tests at 3 months of treatment vs baseline (12.2% vs 61.5%, $P < .001$)	IIB
		NMA of 24 male AGA studies[63]	Topical melatonin showed moderate efficacy (SUCRA = 55.4%), performing above topical procyanidin and topical ketoconazole, but below topical minoxidil	IA
Caffeine 0.2%	Inhibits phosphodiesterase to follicular cell metabolism and proliferation	Open-label trial of 210 male AGA patients treated with topical caffeine 2 mL twice/day vs topical minoxidil 5% 1 mL twice/day for 6 months[116]	Topical caffeine vs topical minoxidil demonstrated similar increase in anagen hairs vs baseline (+11.68% vs +10.59%, $P = .574$)	IIB
Ketoconazole 2%	Antifungal, antiandrogenic, and anti-inflammatory properties	Several RCTs of male AGA patients, largely with combination regimens[119-123]	Topical ketoconazole shampoo contributes to increased hair shaft diameter and hair density	IB
		NMA of 24 male AGA studies[63]	Topical ketoconazole shampoo showed modest efficacy (SUCRA = 30%), performing below topical melatonin and topical procyanidin, but above SPE	IA

AGA, Androgenetic alopecia; *NMA*, network meta-analysis; *RCT*, randomized controlled trial; *SPE*, saw palmetto extract; *SUCRA*, Surface Under the Cumulative Ranking curve.
*Levels of evidence are based on the *Journal of the American Academy of Dermatology* guidelines and reflect study design rather than strength of clinical efficacy: level IA evidence includes evidence from meta-analysis of randomized controlled trials; level IB evidence includes evidence from ≥ 1 randomized controlled trial; level IIA evidence includes evidence from ≥ 1 controlled study without randomization; level IIB evidence includes evidence from ≥ 1 other type of experimental study; level III evidence includes evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies; and level IV evidence includes evidence from expert committee reports or opinions, or clinical experience of respected authorities, or both.

Table V. Adjunctive modalities with the most clinically meaningful evidence in androgenetic alopecia

Adjunct	When it may be considered	Key limitations
Saw palmetto extract	Patient prefers botanical vs prescription antiandrogenic therapy or is unable to tolerate prescription 5ARIs	- Inferior efficacy compared with finasteride - Heterogeneous dosing and formulations
Pumpkin seed oil	Patient prefers botanical vs prescription antiandrogenic therapy or is unable to tolerate prescription 5ARIs	- Limited long-term safety data and small study populations - Heterogeneous dosing and formulations
Rosemary oil	Patient prefers topical botanical options or experiences irritation with topical minoxidil	- Consensus guidelines do not recommend it due to insufficient data
Low-level light therapy	Patient prefers nonpharmacologic, nonsystemic therapy or patient is pregnant or lactating	- Device variability - Lack of standardized treatment protocol
Nonablative fractional laser	Patient interested in procedural adjunct or is unable to tolerate pharmacologic therapies	- Limited long-term data and small study populations - Availability and cost considerations

These interventions may be considered only after optimization of established first-line therapies (eg, topical/oral minoxidil, oral finasteride, spironolactone) and are incorporated based on patient-specific factors, safety considerations, and shared decision-making.
5ARIs, 5-alpha reductase inhibitors.

biological end points, is essential for cross-study comparability. As with all hair loss treatments, including adjuncts, clinical response requires ≥ 6 months, with return to baseline after cessation. Therefore, there is a need for standardized long-term outcome assessment. Mechanistic studies examining inflammatory, oxidative, and microvascular pathways may help identify treatment response biomarkers and guide precision-based therapy selection. Collectively, these efforts support the development of robust, evidence-based guidelines and an individualized, multimodal approach to AGA therapy.

CONCLUSION

In sum, evidence supporting many adjunctive approaches for AGA remains heterogeneous and limited. While FDA-approved therapies remain the only treatments supported by robust, high-quality evidence, adjuncts may offer incremental benefit when used in combination, following a physician-guided, patient-centered framework. Strengthening methodological rigor, improving patient education, and combatting misinformation is essential for refining evidence-based adjunctive AGA management and optimizing long-term outcomes.

Declaration of generative AI and AI-assisted technologies in the writing process

AI was not used in manuscript composition.

Conflicts of interest

Dr Iorizzo has served as a consultant for L'Oreal, Lab. Bailleul, LeoPharma, Johnson & Johnson, Almirall, and Pfizer. Dr Lo Sicco is an investigator for Pfizer; a consultant for Pfizer, Lilly, Priovant, Veradermics, and Ro; and has previously been an investigator for Regen Lab. Dr Tosti has served as a consultant for DS Laboratories, Almirall, Thirty Madison, Eli Lilly, Pfizer, P&G, Myovant, Bristol Myers Squibb, and Ortho-Dermatologics. Dr Shapiro is a consultant for Aclaris Therapeutics, Incyte, and Replicel Life Sciences and has been an investigator for Regen Lab and Pfizer. Dr Lipner has served as a consultant for Moberg Pharmaceuticals and BelleTorus Corporation. Ms Podolsky has no financial disclosures.

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