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Topical Melatonin in Androgenetic Alopecia: Mechanisms of Action and Current Clinical Evidence

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Abstract

Androgenetic alopecia (AGA) is the most common cause of progressive hair loss and significantly affects patients' quality of life. Although established therapies such as minoxidil and finasteride are effective for many individuals, their variable response rates, adverse effects, and adherence challenges have prompted the investigation of complementary treatment options. Melatonin, a hormone best known for regulating circadian rhythms, has emerged as a potential therapeutic agent because of its antioxidant, anti-inflammatory, antiapoptotic, and immunomodulatory properties. The presence of melatonin receptors in the scalp and hair follicles supports its biological role in maintaining follicular homeostasis and promoting hair growth. This narrative review summarizes the mechanisms by which topical melatonin may benefit patients with AGA, including reduction of oxidative stress, prolongation of the anagen phase, modulation of androgen receptor signaling, possible inhibition of 5-alpha reductase activity, and regulation of growth factors involved in follicular regeneration. Available preclinical and clinical studies suggest that topical melatonin can reduce hair shedding, improve hair density, and demonstrate an excellent safety profile, particularly in early-stage disease and as an adjunctive therapy. However, current evidence remains limited by methodological heterogeneity, emphasizing the need for large, well-designed randomized clinical trials to define its efficacy and clinical role.

Keywords: Melatonin, androgenetic alopecia, hair follicle, oxidative stress, anagen phase, topical therapy

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Introduction

Androgenic alopecia (AGA) is the most common form of progressive hair loss and affects both men and women. Its prevalence increases with age and is one of the main causes of dermatological consultation due to its impact on self-esteem, body image and quality of life. The pathophysiology of the disease is complex and results from the interaction between genetic, hormonal and environmental factors that favour the progressive miniaturisation of the hair follicle and the shortening of the anagen phase of the hair cycle. (1–3)

Currently, the treatment of AGA is mainly based on topical minoxidil and oral finasteride, therapies that have demonstrated efficacy in delaying the progres-

sion of the disease and increasing capillary density. However, the clinical response is variable, some patients have adverse effects or poor adherence to treatment, and a significant proportion do not achieve satisfactory results, which has led to the investigation of new therapeutic strategies aimed at complementary pathophysiological mechanisms. (2–4)

Over the past decade, melatonin has gained interest in dermatology for its functions beyond circadian regulation. The skin and hair follicle possess a functional melatoninergic system with MT1 and MT2 receptors, suggesting a role in skin homeostasis, follicular cycle regulation, and inflammatory response. Oxidative stress contributes to the progression of androgenic alopecia by promoting mitochondrial damage, dermal papilla apoptosis, and follicular miniaturization. Thanks to its potent antioxidant and free radical scavenging action, melatonin can protect the hair follicle and promote hair growth. (5–8)

In addition to its antioxidant properties, several experimental studies have shown that melatonin exerts anti-inflammatory, anti-apoptotic and immunomodulatory effects, regulates the expression of cytokines and growth factors, favors the proliferation of keratinocytes and can prolong the anagen phase of the hair cycle. It has also been proposed that it could indirectly modulate the activity of 5α-reductase and androgenic signaling, mechanisms considered fundamental in the pathophysiology of AGA, although this hypothesis still requires further clinical confirmation. (5–8)

The available clinical evidence on topical melatonin remains limited, although published studies show promising results in terms of hair loss reduction, increased hair density, and excellent safety profile, especially when used as an adjunctive treatment in patients with early-stage androgenic alopecia. However, the methodological heterogeneity of the trials, the small sample size, and the scarcity of controlled studies prevent definitive recommendations from being made, so randomized clinical trials with standardized methodologies are still necessary. (4–8)

In this context, the objective of this narrative review is to critically analyze the available scientific evidence on the mechanisms of action of topical melatonin in androgenic alopecia, as well as to synthesize the main experimental and clinical studies that have evaluated its efficacy and safety, in order to define its potential role as a complementary therapy within the comprehensive management of this disease.

Methods

Study design

A narrative review of the literature on the role of topical melatonin in the treatment of androgenic alopecia (AGA) was conducted. The review was designed and reported following the principles of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) statement, insofar as they are applicable to a narrative synthesis. Due to the expected heterogeneity between study designs, populations, interventions, melatonin concentrations, and outcome variables, a meta-analysis was not performed and findings are presented using a structured narrative synthesis.

Bibliographic search strategy

The literature search was carried out in the **PubMed/MEDLINE, Scopus, Web of Science, Embase, and Google Scholar** databases, including publications from January 2000 to December 2025. The search strategy combined MeSH terms and free words using Boolean operators (AND and OR).

The main terms used were:

1. "Melatonin"
2. "Topical melatonin"
3. "Androgenetic alopecia"
4. "Male pattern hair loss"
5. "Female pattern hair loss"
6. "Hair follicle"
7. "Hair growth"
8. "Oxidative stress"
9. "Reactive oxygen species"
10. "Dermal papilla"
11. "Hair cycle"

The search strategy was adapted to the characteristics of each database to maximize the sensitivity and specificity of the identification of relevant studies.

Eligibility Criteria

We included in vitro studies, animal models, randomised clinical trials, observational and prospective studies, systematic reviews, meta-analyses, high-quality narrative reviews and clinical guidelines on androgenic alopecia or topical melatonin. Letters to the editor, conference proceedings, duplicate publications, non-full-text articles, irrelevant studies and papers published in languages other than English or Spanish were excluded.

Study selection

The retrieved titles and abstracts were initially eval-

uated to determine their relevance. Subsequently, the full text of the potentially eligible articles was reviewed. The selection prioritized research that evaluated any of the following aspects:

1. Skin physiology of melatonin;
2. Expression of mt1 and mt2 receptors;
3. Antioxidant mechanisms;
4. Regulation of the follicular cycle;
5. Clinical evidence on topical melatonin;
6. Safety and tolerability;
7. Comparison with conventional treatments for androgenic alopecia.

When there were multiple publications on the same study, the most complete and up-to-date version was used.

Data extraction: Data were systematically collected on author, year, country, design, sample size, population characteristics, type and concentration of melatonin, duration of treatment, efficacy variables, outcomes, adverse events, and conclusions.

Synthesis of evidence: The studies were organized into four categories: physiological mechanisms, experimental and in vitro evidence, clinical studies in androgenic alopecia, and therapeutic implications and future prospects.

Due to the considerable methodological heterogeneity between the included studies, the results were synthesized using a descriptive analysis, emphasizing the consistency of the findings, methodological limitations, and potential clinical applications.

Ethical considerations

As a narrative review based exclusively on previously published scientific literature, this study did not involve the direct participation of humans or animals, so it did not require approval by an ethics committee or the obtaining of informed consent.

Proposed mechanisms of action of melatonin

Oxidative stress

Oxidative stress is one of the most widely accepted pathophysiological mechanisms in the progression of androgenic alopecia. The accumulation of reactive oxygen species induces mitochondrial damage, alters the proliferation of dermal papilla cells, and accelerates the premature entry of the follicle into the catagen phase. Various experimental studies have shown that melatonin acts as a potent antioxidant, decreasing lipid peroxidation, preserving mitochondrial integrity, and increasing the activity of antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase. (5,8,15–17).

Antioxidant protection also seems to contribute to reducing damage induced by ultraviolet radiation and other environmental factors capable of accelerating hair follicle aging. These effects could partially explain the reduction in hair loss observed in various clinical studies on topical melatonin. (8,9,15)

Regulation of the follicular cycle

Various experimental models have shown that melatonin favors the prolongation of the anagen phase and inhibits the premature transition to the catagen phase. A significant increase in hair shaft growth velocity has been observed in human hair follicle cultures after exposure to physiological concentrations of melatonin, an effect that can be blocked by specific antagonists of its receptors, which supports a predominantly receptor-dependent mechanism. (8,11,18)

The prolongation of the growth phase represents one of the most clinically relevant mechanisms, since the follicular miniaturization observed in androgenic alopecia is characterized precisely by a progressive shortening of the anagen and a relative increase in the telogen phase. (3,8,18)

Anti-inflammatory and anti-apoptotic action

Low-grade perifollicular inflammation is currently recognized as an important component of the pathophysiology of androgenic alopecia. Melatonin modulates the production of proinflammatory cytokines, decreases the activation of nuclear factor kappa B (NF-κB), and reduces the expression of inflammatory mediators involved in follicular damage. At the same time, it exerts an anti-apoptotic effect by regulating proteins of the Bcl-2 family and inhibiting caspases, favoring the survival of follicular matrix cells. (8,9,16,19)

Possible interaction with the androgenic pathway

Although the mechanism is not yet fully clarified, several experimental studies suggest that melatonin could indirectly modulate androgen signaling by regulating hormone receptors and enzymes related to androgen metabolism. A possible interaction with 5α-reductase activity and Wnt/β-catenin-associated signaling pathways has also been proposed, although the available clinical evidence remains limited and requires confirmation by controlled trials. (8,17,20)

Preclinical evidence

Preclinical studies support the potential of melatonin in androgenic alopecia. In human hair follicle cultures, it prolonged the anagen phase, stimulated keratinocyte proliferation, and reduced apoptosis by decreasing oxidative stress and preserving mitochondrial function. In animal models, topical application accelerated the transition from telogen to anagen, increased hair thickness, and promoted follicular regeneration. These effects were associated with increased expression of growth factors, such as IGF-1, VEGF, and β-catenin, related to follicular stem cell activation. (17,21–25)

From a molecular point of view, melatonin has also been shown to modulate signaling pathways related to cell survival, including PI3K/Akt, ERK1/2 and Wnt/β-catenin, in addition to decreasing the expression of inflammatory mediators and proinflammatory cytokines. These findings support the hypothesis that melatonin exerts a multifactorial effect on follicular biology and not only an isolated antioxidant action. (17,22,25,26)

Clinical evidence

Clinical evidence on topical melatonin remains limited; however, published studies show consistent results in terms of safety and a potential benefit on reducing hair loss and increasing hair density.

Fischer et al. conducted a double-blind, randomized, placebo-controlled pilot trial in 40 women with androgenetic or diffuse alopecia. A 0.1% topical melatonin solution was applied once daily for 6 months. Among women with androgenetic alopecia (n=12), melatonin significantly increased the anagen hair rate in occipital hair compared with placebo (P=0.012); the study did not report a standardized between-group effect size in the abstract. Plasma melatonin increased during treatment but remained below the physiological night-time peak, and no clinically important systemic safety signal was identified. (27)

Fischer et al. subsequently summarized one pharmacodynamic study and four clinical pre-post studies. In a 35-man androgenetic alopecia cohort treated with topical melatonin, TrichoScan measurements showed mean hair density increasing from 123 hairs/cm² at baseline to 159 hairs/cm² at 3 months and 173 hairs/cm² at 6 months; significant increases were reported in 54.8% and 58.1% of participants at 3 and 6 months, respectively. (8) Other observational cohorts included 30 men and women, 60 patients with hair loss, and a multicenter study of more than 1,800 volunteers, all reporting reductions in hair shedding or perceived severity with good tolerability, although these studies were uncontrolled and used heterogeneous outcome measures. (8)

More recently, Gupta et al. performed a Bayesian network meta-analysis of 15 trials evaluating conventional and non-conventional over-the-counter treatments for male androgenetic alopecia. Melatonin was represented by a single-arm study of 35 men and ranked second among the evaluated topical agents (SUCRA 61.9%), whereas topical minoxidil 5% twice daily ranked first (SUCRA 99.8%). Minoxidil 5% was more effective than topical melatonin for 24-week change in total hair density, with a mean difference of 18.96 hairs/cm² (95% credible interval, 3.51–35.29; P<0.05). These findings support melatonin as a potentially useful adjunct rather than a replacement for established first-line therapy. (17)

Subsequently, several observational studies confirmed that topical administration of melatonin is associated with a significant reduction in the percentage of hairs in the telogen phase and an increase in the proportion of follicles in the anagen phase. These improvements were more evident in patients with mild or moderate androgenic alopecia and in those who initiated treatment in the early stages of the disease. (2,8,27,28)

In women with female pattern alopecia, topical melatonin has also demonstrated a favorable safety profile and a tendency to increase hair density, although the magnitude of the effect continues to vary between different studies. Differences in the concentration used, the duration of treatment, and the methods of evaluation make direct comparison of the results difficult. (2,8,28,29)

Melatonin could be a promising adjunctive therapy in androgenic alopecia. Its combination with minoxidil could generate synergistic effects by combining stimulation of hair growth with the reduction of oxidative stress, inflammation, and follicular damage. However, comparative clinical evidence remains limited and requires higher quality randomized clinical trials. (2,28–30)

Overall, the available studies agree that topical melatonin has an excellent tolerability profile, with a very low incidence of local adverse effects and no relevant systemic alterations (**Table 1**). This characteristic makes it an attractive alternative for patients who have intolerance or contraindications to conventional treatments, as well as for those who require combination therapies aimed at preserving follicular function. (2,8,27–31)

Discussion

The present review shows that topical melatonin represents a promising therapeutic alternative for the management of androgenic alopecia (AGA), due to the fact that it acts on multiple pathophysiological mechanisms involved in follicular miniaturization. Unlike conventional treatments, whose effect is mainly concentrated on peripheral vasodilation or inhibition of androgenic metabolism, melatonin seems to exert a pleiotropic action that includes antioxidant, anti-inflammatory, anti-apoptotic and modulatory activity of the hair follicle cycle. This diversity of mechanisms could explain the favorable results observed in experimental and clinical studies, particularly when used as an adjunctive treatment. (29,32)

One of the most consistent findings is the ability of melatonin to reduce oxidative stress. The excessive production of reactive oxygen species favors mitochondrial damage, apoptosis of dermal papilla cells and shortening of the anagen phase, phenomena closely related to the progression of AGA. Several experimental studies show that melatonin neutralizes free radicals, increases the activity of antioxidant enzymes and protects mitochondrial function, helping to preserve the viability of the hair follicle. These effects could constitute one of the main mechanisms responsible for the reduction of hair loss observed in the available clinical studies. (33)

Another relevant aspect is the influence of melatonin on the follicular cycle. Experimental evidence indicates that it prolongs the anagen phase and delays the transition to the catagen phase, favoring a longer hair growth time. This mechanism is especially important in AGA, where the progressive reduction of the duration of the anagen is one of the most characteristic pathophysiological alterations. Although studies in humans are limited, the available results show increases in hair density, hair shaft thickness and the proportion of hairs in the anagen phase after topical application of melatonin. (34)

Clinical evidence indicates that topical melatonin has a favorable safety profile, with mild and transient adverse events, mainly local irritation or pruritus, without relevant systemic effects. Although it cannot yet be recommended as a first-line treatment for androgenic alopecia, it is an attractive alternative for patients with intolerance to conventional therapies. Compared to minoxidil and finasteride, which continue to be the most evidenced treatments, melatonin could play a role as an adjuvant therapy thanks to complementary mechanisms. However, robust clinical trials are required to confirm its efficacy and define its use in combination. (34,35)

Despite the encouraging results, the methodological quality of the available evidence remains an important limitation. The studies are markedly heterogeneous in terms of design, sample size, diagnostic criteria, melatonin concentrations used, duration of treatment, and endpoints. In addition, most correspond to pilot studies or prospective series with relatively short follow-up, which makes it difficult to draw firm conclusions about the true magnitude of the clinical benefit and its long-term persistence. (29)

Another important limitation lies in the absence of standardized protocols for the topical formulation of melatonin. The concentrations used in the literature vary between 0.0033% and 0.1%, using different pharmaceutical vehicles and application frequencies. This variability makes it difficult to compare between studies and limits the reproducibility of the results. In addition, there is still no consensus regarding the optimal concentration, the ideal duration of treatment, or the subgroups of patients that could benefit the most. (29,33)

The future prospects are promising. The development of new formulations using nanostructured systems, liposomes and controlled-release vehicles could improve follicular penetration and increase the bioavailability of topical melatonin. At the same time, the identification of molecular biomarkers would make it possible to select patients with the highest probability of therapeutic response and move towards personalized medicine strategies. However, these approaches will need to be confirmed through multicenter, randomized, long-term follow-up clinical trials. (34)

Overall, the available scientific evidence supports the biological plausibility of the use of topical melatonin in androgenic alopecia and demonstrates a favorable safety profile. However, the strength of the clinical evidence remains moderate due to the limited methodological quality of the available studies. Consequently, melatonin should currently be considered a complementary therapy with clinical potential, whose definitive role will depend on future high-quality controlled studies that allow its efficacy, indications and position within the therapeutic algorithm of androgenic alopecia to be precisely defined. (35)

Table 1. Summary of key clinical evidence for topical melatonin in androgenetic alopecia

Study	Design/sample	Melatonin regimen	Duration	Main outcome	Safety/limitations
Fischer et al., 2004 (27)	Double-blind RCT; n=40 women (AGA n=12; diffuse alopecia n=28)	0.1% topical solution, once daily	6 months	AGA subgroup: significant increase in occipital anagen hair rate vs placebo (P=0.012).	No important systemic signal; pilot study and small AGA subgroup.
Fischer et al., 2012 (8)	Pharmacodynamic study + 4 clinical pre-post studies; key AGA cohort n=35 men	Topical melatonin solution, once daily	3–6 months	35-man cohort: hair density 123→159 hairs/cm² at 3 months and 123→173 hairs/cm² at 6 months; significant increases in 54.8% and 58.1%, respectively.	Uncontrolled observational data; heterogeneous endpoints and populations.
Fischer et al., 2012 observational cohorts (8)	Observational cohorts: 30 men/women; 60 patients; multicenter >1,800 volunteers	Topical melatonin solution	30 days-3 months	Reduced perceived severity/hair shedding; multicenter hair-pull test improved substantially.	No placebo/control groups; subjective and heterogeneous measures.
Gupta et al., 2025 (17)	Bayesian network meta-analysis; 15 trials; melatonin represented by a single-arm n=35 study	Topical melatonin	24 weeks	Melatonin SUCRA 61.9%; minoxidil 5% SUCRA 99.8%. Minoxidil 5% vs melatonin: MD 18.96 hairs/cm² (95% CrI 3.51–35.29).	Indirect comparison; melatonin evidence based on a single-arm trial.

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