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ORAL AND TOPICAL FINASTERIDE IN THE TREATMENT OF MALE ANDROGENETIC ALOPECIA – NARRATIVE REVIEW

Ewa Otręba[1] ✉, ***Julia Wendt[1]***,
Michał Hebel[2], ***Szymon Stankowski[1]***,
Mikołaj Szewczykowski[1], ***Tomasz Klinkosz[1]***,
Szymon Korczyk[3], ***Dagmara Fredrich[3]***,
Wiktor Fredrich[3]

[1]F. Ceynowa Specialist Hospital in Wejherowo, Poland
[2]PCK Maritime Hospital in Gdynia, Poland
[3]Medical University of Gdańsk, Poland

✉ ***otreba.ewa@icloud.com***

ABSTRACT

BACKGROUND

Androgenetic alopecia is the most common form of progressive non scarring hair loss in men and is driven by dihydrotestosterone mediated miniaturization of hair follicles. The condition may negatively affect psychological well being and quality of life. Finasteride, a 5 alpha reductase inhibitor, is a key pharmacologic therapy.

AIMS

To evaluate the mechanism of action, metabolism, efficacy, and safety of finasteride in male androgenetic alopecia, with comparison of oral and topical formulations, assessment of systemic exposure and DHT reduction, and review of adverse effects, alternative FDA approved treatments, combination strategies, and broader clinical relevance.

METHODS

A narrative review was conducted using PubMed and Google Scholar for English language studies published between 2015 and 2026. Fifty one references were included. Priority was given to randomized controlled trials, systematic reviews, meta analyses, pharmacokinetic studies, and pharmacovigilance reports. Data were synthesized descriptively without formal quality assessment or meta analytic pooling.

RESULTS

Finasteride inhibits type II and III 5 alpha reductase, lowering scalp and serum DHT levels and stabilizing hair growth. Oral finasteride 1 mg daily significantly improved hair count and density, with reported increases of 18.37 hairs per cm² in comparative analyses. Topical 0.25 percent formulations showed comparable efficacy after 24 weeks with markedly lower systemic exposure and smaller reductions in serum DHT.

Oral therapy has been associated with sexual adverse effects and reported neuropsychiatric symptoms. Post

finasteride syndrome remains controversial, and pharmacovigilance signals regarding depression and suicidal ideation require cautious interpretation. Combination regimens, particularly with topical minoxidil, demonstrated enhanced outcomes in selected studies. Finasteride is also approved for benign prostatic hyperplasia and has shown a reduction in overall prostate cancer incidence in long term trials.

CONCLUSIONS

Finasteride is a major therapeutic option for male androgenetic alopecia. Oral therapy provides robust efficacy but greater systemic exposure, whereas topical formulations offer similar hair growth outcomes with reduced systemic DHT suppression. Given the narrative design and heterogeneity of available data, conclusions should be interpreted with caution.

Keywords: androgenetic alopecia, finasteride, 5 alpha reductase inhibitors, topical therapy, post finasteride syndrome

INTRODUCTION

Androgenetic alopecia (AGA), also known as male pattern baldness, is the most frequent form of non-scarring hair loss in the population. This condition affects 30-50% of men before the age of 50. Some studies indicate that AGA can negatively influence how patients perceive their appearance, potentially increasing anxiety and contributing to depression. [1,2] However, some recent studies found no significant impact of AGA on depression levels, and only a moderate effect on quality of life. [3]

AGA is also a proven factor for coronary heart disease, prostate cancer, and metabolic syndrome. [4] A familial predisposition to this condition has been proven, as well as racial differences. Japanese, Chinese, and African Americans are less likely to be affected than Caucasians. [5] Hereditary factors are responsible for 80% of the cases. [1]

The etiopathogenesis of AGA is multifactorial. Multiple studies have clearly identified two main genetic risk loci of androgenetic alopecia: the AR/EDA2R locus on chromosome X and the 20p11 locus on chromosome 20. [5] In addition to genetic causes, it also includes hormonal and environmental components. Key factors in the pathophysiology of this condition are changes in the hair cycle, miniaturization of hair follicles, and inflammation. [1,2,6]

Androgenetic alopecia in men is characterized by gradual thinning of the hair, affecting mostly the top of the head and the front of the scalp. This distinctive pattern of hair loss is a result of the distribution of androgen receptors in the scalp. [7] AGA results from androgen-dependent miniaturization of the hair follicles on the scalp, one of the causes of which is the presence of DHT in this area. Finasteride, a type II 5α-reductase inhibitor, reduces DHT levels in serum and scalp tissue by inhibiting the conversion of testosterone to DHT. [8] Each hair is developed in a hair follicle, which goes through three different phases divided into the anagen phase (growing phase), the catagen phase (transition phase), and the telogen phase (resting phase). Before the end of the telogen stage, the hair follicle re-enters the anagen stage and begins to produce a new hair even before the previous one is shed. In exceptional cases, the hair follicle may go into a dormant period, during which it remains empty for a certain amount of time (kenogen phase). In AGA, the anagen phase shortens with each cycle, while the duration of the telogen phase remains constant or elongates. Eventually, the anagen phase becomes so abbreviated that the growing hair is unable to reach sufficient length to reach the skin surface, leaving an empty hair follicle. [1,2]

Patients with AGA have been observed to have higher levels of DHT, which is responsible for the development and maintenance of male sexual characteristics, and type II 5-alpha reductase, which is responsible for converting testosterone into DHT in the outer layer of the hair follicle. Although testosterone levels are similar in people with and without AGA, levels of unbound testosterone, an active form, are higher in people with AGA. Furthermore, individuals who were castrated before puberty, those with complete androgen insensitivity syndrome (CAIS), or those with type II 5-alpha-reductase deficiency have a lower incidence of AGA, providing further evidence that androgens are fundamental to the development of AGA. [7]

AIMS

Androgenetic alopecia (AGA) is a condition that affects a substantial proportion of adult men, negatively influencing self-esteem and psychological well-being, and often impairing daily functioning. This review of current scientific literature aims to analyze the therapeutic outcomes of finasteride in the treatment of AGA in men, comparing its oral and topical formulations. A clearer understanding and comprehensive presentation of the effects of finasteride will assist practicing clinicians in selecting appropriate therapeutic strategies for patients with AGA, while also increasing awareness of potential adverse effects associated with treatment. The literature review also mentions modern combination methods and future molecular targets that may revolutionize the fight against hair loss.

RESEARCH OBJECTIVES

- Description of the mechanism of action and metabolism of finasteride in the treatment of AGA.
- Analysis and comparison of the therapeutic outcomes of oral and topical finasteride in men, including systemic exposure and the reduction of DHT levels.
- Enhancement of awareness of potential adverse effects, with particular reference to Post-Finasteride Syndrome, depression, and suicide risk.
- Comparison of FDA-approved treatment options for AGA.
- Review of modern combination therapies that are considered the most promising treatment strategies.
- Identification of future molecular targets and regenerative-based therapies that may revolutionize hair loss treatment.
- Discuss the broader clinical relevance of finasteride, including its FDA-approved use for benign prostatic hyperplasia (BPH) and its role in prostate cancer prevention.

METHODS

This study was conducted as a narrative review of the current scientific literature evaluating the role of finasteride in the treatment of male androgenetic alopecia .

LITERATURE SOURCES AND SEARCH STRATEGY

A structured literature search was performed using the electronic databases PubMed and Google Scholar. The search included publications available in English between 2015 and 2026. Selected earlier key publications were additionally included when necessary to describe the pathophysiology of androgenetic alopecia or the pharmacological profile of finasteride.

The search was conducted using combinations of the following keywords: androgenetic alopecia, hair loss, 5 alpha reductase inhibitors, finasteride, topical finasteride, oral finasteride, post-finasteride syndrome, depression, suicide risk, benign prostatic hyperplasia, prostate cancer prevention. Boolean operators were applied to combine search terms.

STUDY SELECTION

Publications were screened for relevance to the predefined objectives of the review. Priority was given to randomized controlled trials, phase I and phase III clinical studies, systematic reviews, meta-analyses, pharmacokinetic studies, pharmacovigilance analyses, and clinical guidelines concerning male androgenetic alopecia and finasteride therapy.

A total of 51 references were included in the final analysis.

DATA EXTRACTION AND SYNTHESIS

Data were extracted manually from the selected publications. Extracted variables included study design, population characteristics, dosage and route of finasteride administration, changes in hair count and density, serum and scalp dihydrotestosterone levels, systemic exposure, and reported adverse events. Additional data concerning psychiatric safety signals, post-finasteride syndrome, and regulatory safety reviews were also analyzed.

The synthesis of results was descriptive. A comparative analysis was performed between oral finasteride 1 mg daily and topical formulations, most commonly 0.25 percent solutions or sprays. The efficacy and safety profiles were also discussed in relation to other FDA-approved treatments for androgenetic alopecia.

Given the narrative design of the review, no formal risk of bias assessment or quantitative meta-analysis was performed.

RESULTS

MECHANISM OF ACTION

Finasteride is a competitive inhibitor of type II and III 5-alpha reductase isoenzymes, suppressing the conversion of testosterone to DHT - the main pathogenic androgen in AGA. [9,10] DHT shows approximately 5 times greater affinity for androgen receptors than testosterone. [2] Finasteride shows minimal selectivity for type I 5-alpha reductase, which is found in sebaceous glands, skin papilla cells, sweat glands, keratinocytes of the epidermis, and hair follicles. Type II occurs in the outer sheaths of hair follicles, vas deferens, seminal vesicles, prostate, as well as

epididymides, and its inhibition significantly reduces DHT levels in both serum and scalp. [11,12] This translates into a decrease in its effect on androgen receptors in hair follicles and prevents their miniaturization. [13] By blocking the formation of DHT, finasteride restores the normal length of the anagen phase, stabilizes hair loss, and stimulates hair regrowth. [10] When administered at a dose of 1 mg orally per day, finasteride can reduce DHT levels in serum and scalp by up to 70%. [14] After discontinuing treatment, DHT levels return to normal within approximately 14 days. [15] Maintaining therapeutic effects depends on continuous use of the drug. Discontinuation of therapy results in renewed progression of hair loss within approximately 12 months. [16]

METABOLISM

Finasteride is extensively metabolized in the liver by the CYP3A4 enzyme system into two active metabolites retaining approximately 20% of the 5-alpha reductase inhibitory activity. [9,11] Medicine has an oral bioavailability of approximately 65% in healthy male subjects, meaning about two-thirds of an oral dose is absorbed into the bloodstream. [9] This absorption is not affected by food, and maximum plasma concentrations are reached within 1 to 2 hours after taking a dose. Finasteride is bound to plasma proteins by approximately 90%. [11] The half-life for elimination varies depending on age. In men aged 18–60, it is approximately 5–6 hours. In older males, over 70 years of age, it may be extended to 8 hours. [17] Multiple-dose administration of topical finasteride ensures a stable half-life, minimal change in clearance rate, and good tolerability. [18] It is excreted with about 57% in feces and 39% in urine. [12]

EFFECTS AND ROUTES OF ADMINISTRATION

Clinical trials conducted over the past three decades have consistently shown the efficacy of finasteride. [19] These studies indicate benefits such as clinically significant increases in hair count, hair weight, and global photographic assessment scores with a daily dose of 1 mg finasteride compared to placebo. [19] Among the treatments evaluated, daily administration of 1 mg finasteride yielded the highest increase in hair count (18.37 hairs/cm²), relative to low-level laser therapy (LLLT) (17.66 hairs/cm²), 5% minoxidil twice daily (14.94 hairs/cm²), and 2% minoxidil twice daily (8.11 hairs/cm²). [12] Noticeable effects of treatment can be seen after 6 months, but it may take up to 12 months to become evident in certain individuals. [5]

Modern Bayesian analyses indicate that finasteride (1 mg/day) shows the strongest hair growth during the first 12 weeks, followed by stagnation or a resistance phase. In studies conducted in 2023, finasteride performed worse in terms of growth continuity compared to newer multi-component preparations (e.g. ALRV5XR), which did not show resistance after 12 weeks. [24]

Topical finasteride shows efficacy comparable to oral finasteride 1 mg daily while being associated with fewer sexual adverse events, making it a potential alternative for patients concerned about systemic effects. [20] Following 24 weeks of treatment, 0.25% topical finasteride significantly increased hair density in comparison with placebo. [19]

In the study by A. Rossi et al. (2023), the researchers evaluated three groups of men with AGA in a 6-month, prospective, randomized, assessor-blinded trial. The group treated with 0.25% topical finasteride exhibited an increase in hair density over the course of the study; however, statistical significance was observed only in the group receiving combination therapy with 0.25% topical finasteride and 5% topical minoxidil. [21]

M. Caserini et al. (2016) investigated the efficacy of a novel 0.25% topical finasteride formulation administered once daily in inhibiting scalp DHT. The results demonstrated that the 0.25% finasteride solution effectively reduced scalp DHT levels, which may minimize unwanted sexual adverse effects associated with systemic DHT suppression. [22]

In a major randomized phase III study, the efficacy of topical finasteride (0.25% spray solution) was numerically similar in increasing the number of hairs on the targeted area after 24 weeks to that of oral finasteride (1 mg) and significantly superior to placebo. [23] The main goal of topical finasteride use is to achieve high efficacy with minimal risk of side effects associated with systemic DHT blockade.

A direct comparison of oral and topical finasteride in terms of systemic exposure, DHT suppression, hormonal effects, and adverse events is presented in Table 1.

Table 1. Comparison of oral and topical finasteride in the treatment of androgenetic alopecia.

	Oral finasteride (1 mg/ day)	**Topical finasteride (0.25% solution)**

Systemic exposure	High exposure [25]	Significantly reduced; maximum average plasma concentrations of the drug are over 100 times lower than with the oral preparation [25]
Reduction of serum DHT	Reduces serum DHT concentration by approximately 55.6% after 24 weeks [25]	Reduces serum DHT levels by approximately 34.6% after 24 weeks [25]
DHT level in the scalp	Effectively reduces DHT concentration in the scalp [16]	Research suggests that daily application once a day may be more effective than oral 1 mg [16]
Plasma testosterone level	No major changes are usually observed [16]	No significant changes are observed [16]

SIDE EFFECTS

The most commonly reported side effects related to topical finasteride therapy may include skin erythema, increased liver enzyme levels, nocturnal enuresis, testicular pain, headaches, presyncope, oropharyngeal pain, and contact dermatitis. [8,26] Oral finasteride has been associated with adverse effects, including orthostatic hypotension and dizziness. [8] In addition to its inhibition of 5-alpha reductase, finasteride may influence epinephrine synthesis through binding to and inhibition of phenylethanolamine N-methyltransferase. This mechanism may contribute to sexual and psychological adverse effects, including reduced libido, erectile dysfunction, and ejaculatory disorders. [15,16,27]

Another reported, though inconclusive, effect relates to impaired fertility. Some studies have observed a reduction in sperm count and altered spermatogenesis during finasteride treatment, whereas others reported no significant changes in men receiving 1 mg of finasteride daily. Notably, the decrease in sperm count was reversible and resolved within 3–4 months following treatment discontinuation. [8]

Some studies have reported a significant increase in depressive symptoms and suicidal ideation associated with oral finasteride use in recent years. [28–30]

Given the potential for alarming symptoms during finasteride therapy, it is crucial that patients are properly informed about possible adverse effects, and prescribing clinicians should perform a comprehensive psychiatric evaluation before initiation, with continued monitoring of suicide risk during the course of treatment. [8,10,31,32]

POST-FINASTERIDE SYNDROME (PFS)

PFS, first suggested in 2012, is a set of persistent symptoms occurring during and/or after discontinuation of finasteride therapy, both oral and topical. The side effects include significant and permanent sexual and neuropsychiatric symptoms, such as loss/reduced libido, erectile and ejaculatory dysfunction, depression, anxiety, panic attacks, and suicidal ideation. [9,33,34] However, there is no general consensus that finasteride is responsible for these side effects. The studies do not take contributing risk factors into consideration, the symptoms are non-specific and frequently seen in the general population, and AGA on its own can be associated with a higher risk for depression and anxiety. [33] Despite the increasing number of reported adverse effects among patients, PFS is still not formally recognized by the medical community. There are also currently no guidelines on how to manage the treatment of PFS. [34]

REPORTS OF DEPRESSION AND SUICIDE RISK

Data from the FAERS database show a clear upward trend in the number of reports of suicidal tendencies, which peaked in 2024. Most reports come from young men (aged 18–40) using the hair loss medication. In 2024, the European Medicines Agency (EMA) initiated a new safety review of products containing finasteride to better understand the link between the drug and suicidal thoughts. [35] Some analyses suggest that the disproportionately high number of adverse event reports for finasteride (compared to the stronger dutasteride) may be due to media coverage of post-finasteride syndrome (PFS) and the nocebo effect, rather than solely to the direct pharmacological action of the drug. [36]

DISCUSSION

As the main goal of this review was to collect the available data about the role of finasteride in the treatment of AGA,

we would like to compare now the main advantages and disadvantages of both oral and topical application of finasteride and place this substance usage next to the others, approved and recommended by world-renowned organizations such as the Food and Drug Administration.

POSITION OF THE FOOD AND DRUG ADMINISTRATION (FDA)

The FDA has approved two main treatments for androgenetic alopecia: topical minoxidil and oral finasteride. Minoxidil is a treatment for both men and women, while finasteride is specifically approved for men to reduce the hormone DHT, which is a primary cause of AGA. [16] Minoxidil works by increasing blood flow to the hair follicles and lengthening the growth phase, while finasteride works by blocking the conversion of testosterone to DHT, a hormone that shrinks hair follicles. [12] In addition to these medications, some devices using low-level laser therapy (LLLT) have also received FDA clearance for treating this condition in both men and women. [37]

For clarity and structured comparison of FDA approved therapeutic options for androgenetic alopecia, the key characteristics of finasteride, minoxidil, and low level laser therapy are summarized in Table 2.

Table 2. Comparison of FDA-approved AGA treatment methods

Treatment applied	Main indications	Recommended use	Mechanism of action	Key adverse events
Finasteride (oral)	Men (AGA) [16]	1 mg once daily [11]	A type II 5-alpha reductase inhibitor that blocks the conversion of testosterone to dihydrotestosterone. [16]	Sexual dysfunction (decreased libido, erectile/ ejaculatory dysfunction), gynecomastia, mood disorders (depression, suicidal thoughts) [11,15]
Minoxidil (topical)	Men and women (AGA, female pattern hair loss) [16]	2% or 5% solution/foam; most commonly 1 ml twice daily for men [38]	Vasodilation, induces cell proliferation, prolongs the anagen phase [13]	Scalp irritation, itching, contact dermatitis, hypertrichosis [8]
Low-Level Laser Therapy (LLLT)	Men and women (AGA) [15]	Devices for home use (such as combs, hats); usually 3 times a week or daily. [37]	Photobiomodulation stimulates mitochondria, leading to increased production of ATP and growth factors, prolongs the anagen phase. [39]	Minimal and mild: scalp tenderness/ irritation, itching, paresthesia. [8]

EFFICACY AND ADMINISTRATION ROUTES

The compiled evidence supports the efficacy of a 1mg/day oral dosage of finasteride, demonstrating clinically significant benefits, including both hair count and hair weight, compared to placebo. Notably, this dose yielded the highest increase in hair count among the evaluated treatments, surpassing LLLT and both 5% and 2% minoxidil [19].

Another significant development in the management of AGA is the emergence of topical finasteride. Comparative studies suggest that 0.25% topical finasteride offers efficacy numerically similar to the 1mg oral dose after 24 weeks of treatment, simultaneously being substantially superior to placebo. [21]

The major advantage of the topical formulation is its reduced systemic exposure, with plasma concentration over 100 times lower than the oral application. [16] While oral formulation reduces serum DHT by approximately 56.6% after 24 weeks, the topical solution achieves a clearly lower reduction of approximately 34.6%, suggesting that the topical local inhibition in the scalp is sufficient for therapeutic benefit while minimizing systemic suppression. [25]

Furthermore, some studies suggest that topical application may be more effective in reducing local dihydrotestosterone (DHT) levels than oral administration, which may be associated with a lower incidence of treatment-related sexual adverse effects while maintaining comparable therapeutic efficacy. [25,40,41] Topical finasteride can also serve as an effective maintenance therapy to preserve hair density after initial improvement with oral finasteride, thereby avoiding the need for continuous systemic medication. [8]

The review highlights the possible potential of dual therapy combining 1mg oral finasteride with 2% topical minoxidil, which presents the most desirable outcome. [5,15]

CG2001 foam is a new, isopropyl alcohol-free formulation combining minoxidil (5%) and finasteride (0.025%–0.1%). Studies from 2026 show that systemic exposure to finasteride after use is more than 30 times lower than after a 1 mg tablet, making it a promising and safer alternative. [42]

DUAL CLINICAL RELEVANCE AND UNDESIRABLE EFFECTS

In addition to treating AGA, oral finasteride at a dose of 5 mg per day was approved by the FDA in 1992 for the treatment of benign prostatic hyperplasia (BPH). Our review confirms an established role of finasteride in reducing the prostate volume and alleviating lower urinary tract symptoms. [43] DHT is the main pathogenic androgen involved in the pathogenesis of BPH, and a decrease in its level results in a reduction in prostate volume. [23] This translates into an improvement in symptoms of the lower urinary tract resulting from compression of the urethra caused by prostate enlargement. [14] Long-term treatment with finasteride significantly reduces the incidence of acute urinary retention and diminishes the need for surgical intervention in the treatment of BPH, with a 55-57% risk reduction over a 4-year period. [44] Clinical trials such as the Prostate Cancer Prevention Trial have investigated the potential of finasteride as a chemopreventive agent in the prevention of prostate cancer. [14] The trial used a dose of 5 mg per day. It showed a reduction in the overall risk of developing prostate cancer of approximately 24.8% over 7 years compared to placebo. [45]

Despite the therapeutic benefits, it is crucial to acknowledge the potential for undesired effects. Oral finasteride has been associated with sexual and psychological side effects, including reduced libido, erectile and ejaculatory dysfunction. [15,16,27]

A major area of controversy is the PFS, a cluster of sexual and psychiatric symptoms reported during and after discontinuation of finasteride therapy. These symptoms include permanent and significant loss of libido, sexual dysfunctions, depression, anxiety, panic attacks, and suicidal ideation. [9,33,34] However, there is currently no general medical consensus on finasteride being solely responsible for PFS, as the symptoms are non-specific and AGA is itself linked to an increase in anxiety and depression risks. [33] Given the severity of potential symptoms, patients must be thoroughly informed about the possible adverse effects prior to starting therapy.

COMBINATION TREATMENT

In light of available sources, combination therapies are considered a more effective treatment strategy for androgenetic alopecia (AGA) than monotherapy, as they allow for simultaneous action on various pathogenic mechanisms of the disease. [46] The best results are achieved by synergistically combining FDA-approved drugs with modern supportive methods such as microneedling or laser therapy. [7,8]

A retrospective real-world analysis of over 600,000 patients showed that 80.4% of men using innovative combinations of topical finasteride and minoxidil are satisfied with the treatment, and less than 3% of respondents report side effects. [47]

The latest network meta-analysis (2026) showed that multi-ingredient supplements such as Nutrafol and ALRV5XR are the most effective natural products for improving hair density. They can serve as a beneficial supplement or alternative to conventional treatments. [48]

THE FUTURE OF TREATMENT – NEW MOLECULAR TARGETS

Research from 2025–2026 identifies new potential targets for AGA treatment, such as the SOD1 and KL (Klotho) genes, which may become targets for a new generation of drugs that bypass the androgen pathway and the associated side effects of finasteride. [49]

Fibroblast growth factor 7 (FGF-7) has been identified as a key mediator promoting the anagen phase and stem cell activation. Modern approaches suggest the use of mRNA or exosomes for the precise delivery of FGF-7 to hair follicles. [50]

LIMITATIONS OF THE REVIEW AND FUTURE DIRECTIONS

The major limitations of this review stem from the nature of the included literature, particularly the ongoing debate about PFS and the future need for more comparative data.

Invasive methods of AGA treatment, such as platelet-rich plasma use, seem to be safe and effective alternatives for pharmaceutical therapy. Some researchers believe that the future will be based exclusively on regenerative-based therapies [51].

CONCLUSIONS

The analysis of the reviewed publications indicates that finasteride acts as an inhibitor of type II and III 5 alpha reductase isoenzymes, leading to reduced formation of dihydrotestosterone and attenuation of androgen dependent miniaturization of hair follicles in androgenetic alopecia. The drug is metabolized in the liver via the CYP3A4 system and requires long term administration to maintain therapeutic efficacy.

Available evidence suggests that the oral form at a dose of 1 mg daily provides a significant increase in hair density and a marked reduction in serum DHT levels. Topical formulations at a concentration of 0.25 percent demonstrate comparable clinical outcomes with lower systemic exposure and a less pronounced decrease in circulating DHT. These differences may be relevant when selecting therapy based on the individual risk profile of the patient.

Adverse events, including sexual dysfunction and possible neuropsychiatric symptoms, have been described in clinical studies and pharmacovigilance reports. Post finasteride syndrome remains a subject of scientific debate, and current data do not allow definitive conclusions regarding causal relationships or the true incidence of persistent symptoms.

In comparison with other FDA approved treatments for androgenetic alopecia, such as minoxidil and low level laser therapy, finasteride represents one of the principal therapeutic options. Combination regimens, particularly with minoxidil, are considered potentially more effective, although their advantages require confirmation in studies with comparable methodological design.

Beyond its role in alopecia, finasteride is used in the management of benign prostatic hyperplasia and has been investigated in the context of prostate cancer risk reduction, reflecting its broader clinical relevance.

Future directions include the identification of novel molecular targets and the development of regenerative based strategies; however, their clinical applicability remains under investigation.

It should be emphasized that these conclusions are based on a narrative analysis of the available literature without formal quality assessment or quantitative meta analytic synthesis, which limits the level of evidence and necessitates cautious interpretation of the summarized findings.

AUTHORS' CONTRIBUTIONS

Conceptualization: Ewa Otręba, Julia Wendt, Michał Hebel

Methodology: Julia Wendt, Ewa Otręba, Michał Hebel

Investigation and data collection: Mikołaj Szewczykowski, Tomasz Klinkosz, Szymon Korczyk, Ewa Otręba, Julia Wendt, Michał Hebel, Szymon Stankowski, Dagmara Fredrich, Wiktor Fredrich

Literature review and data extraction: Mikołaj Szewczykowski, Tomasz Klinkosz, Szymon Korczyk, Ewa Otręba, Julia Wendt, Michał Hebel, Szymon Stankowski, Dagmara Fredrich, Wiktor Fredrich

Writing – original draft: Szymon Stankowski, Dagmara Fredrich, Wiktor Fredrich, Ewa Otręba, Michał Hebel, Julia Wendt, Szymon Korczyk, Mikołaj Szewczykowski, Tomasz Klinkosz

Writing - tables preparation: Julia Wendt

Writing – review and editing: Julia Wendt, Ewa Otręba, Michał Hebel, Szymon Stankowski, Mikołaj Szewczykowski, Tomasz Klinkosz, Szymon Korczyk, Dagmara Fredrich, Wiktor Fredrich

Final manuscript formatting in accordance with journal guidelines: Julia Wendt, Ewa Otręba

All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

USE OF AI

During the preparation of this manuscript, the authors used AI tools to assist with grammatical error correction, synonym selection, and sentence paraphrasing to enhance clarity and appropriate academic writing style. All AI-generated suggestions were critically evaluated by the authors, and only appropriate ones were adopted into the manuscript.

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CONFLICT OF INTEREST

Authors declare no conflicts of interest.

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