



Pharmacological Role and Clinical Applications of 5 α -Reductase Inhibitors-Review Article for Pharmacists and Healthcare Professionals

Saud Alhabeeb¹, Abdullah Abdulaziz Almetrek¹, Marwan Ibraheem Al-Amri¹, Fahad Abdullah Saleh Alfahaid², Abdullah Abdulaziz Abdulrahman Alsheeb², Ali Abdulrahman Ali Alajlan², Mohanad Khaled Ibrahim Alsaudi²

¹ Kingdom of Saudi Arabia, Red Crescent Authority.

² Kingdom of Saudi Arabia, Qassim University.

Abstract

Background: 5 α -reductase inhibitors (5-ARIs), including finasteride and dutasteride, are FDA-approved for treating benign prostatic hyperplasia (BPH) and androgenic alopecia. These agents inhibit the conversion of testosterone to dihydrotestosterone (DHT), reducing prostate volume in BPH and slowing hair loss in androgenic alopecia. Despite their efficacy, concerns regarding adverse effects, such as sexual dysfunction and post-finasteride syndrome, necessitate careful patient selection and monitoring.

Aim: This review evaluates the pharmacological mechanisms, clinical applications, and safety profiles of 5-ARIs, providing evidence-based guidance for pharmacists and healthcare professionals.

Methods: A comprehensive literature review was conducted, analyzing clinical trials, meta-analyses, and guidelines on 5-ARI use in BPH, androgenic alopecia, and off-label indications.

Results: 5-ARIs significantly improve urinary symptoms in BPH (reducing prostate volume by ~25%) and stabilize hair loss in 88% of alopecia patients. Dutasteride offers broader DHT suppression but clinical efficacy similar to finasteride. Adverse effects include sexual dysfunction (e.g., erectile dysfunction, reduced libido) and gynecomastia. Off-label uses (e.g., hirsutism, bladder cancer prevention) show promise but require further validation.

Conclusion: 5-ARIs are cornerstone therapies for BPH and androgenic alopecia, though their use demands individualized risk-benefit assessment. Multidisciplinary collaboration ensures optimal outcomes.

Keywords: 5 α -reductase inhibitors, finasteride, dutasteride, BPH, androgenic alopecia, DHT inhibition

Introduction

5 α -reductase inhibitors (5-ARIs), such as finasteride and dutasteride, are clinically approved by the U.S. Food and Drug Administration (FDA) for the treatment of two primary conditions: benign prostatic hyperplasia (BPH) and androgenic alopecia, also known as male pattern hair loss [1][2]. These agents act by inhibiting the enzyme 5 α -reductase, which is responsible for converting testosterone into its more potent form, dihydrotestosterone (DHT). The therapeutic use of 5-ARIs in BPH is aimed at reducing prostate volume, improving lower urinary tract symptoms, and decreasing the risk of acute urinary retention and the need for surgical intervention. In androgenic alopecia, these

medications help to slow the progression of hair loss and, in some cases, promote partial regrowth by minimizing the effects of DHT on hair follicles. Finasteride selectively inhibits type II 5 α -reductase, while dutasteride inhibits both type I and type II isoenzymes, resulting in a broader suppression of DHT synthesis. This pharmacological distinction explains differences in efficacy and side effect profiles observed between the two agents. Dutasteride has been shown to achieve more significant DHT suppression in both the serum and the scalp, which may contribute to its enhanced clinical outcomes in some cases. However, both agents are considered effective and are used according to patient-specific factors, including tolerability,

comorbidities, and treatment goals. The application of 5-ARIs is based on evidence demonstrating their impact on hormonal pathways associated with prostatic enlargement and follicular miniaturization. Their use requires consideration of potential adverse effects, particularly those related to sexual function and hormonal balance, and is generally reserved for patients in whom the benefits outweigh these risks.

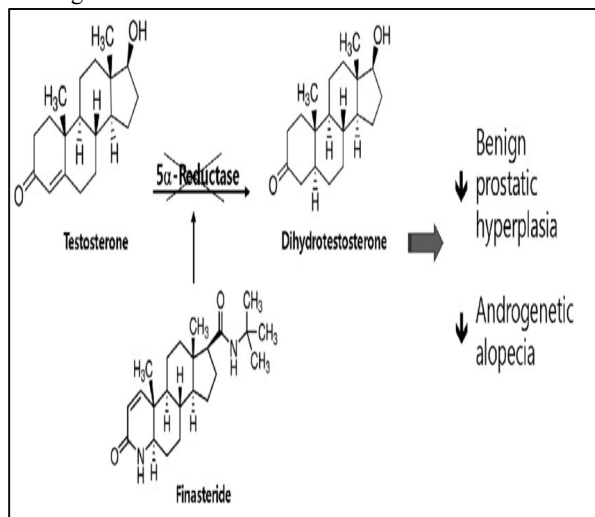


Figure-1: Activators and Inhibitors of 5 α -Reductase Enzyme.

FDA-Approved Indications Benign Prostatic Hyperplasia

Benign prostatic hyperplasia (BPH) is a progressive, nonmalignant enlargement of the prostate gland commonly observed in aging males. Its prevalence correlates positively with age, affecting a growing portion of the male population over time. Clinically, BPH is associated with a range of lower urinary tract symptoms (LUTS) that can significantly impair a patient's daily functioning and quality of life. These symptoms include nocturia, urinary urgency, increased frequency of urination, a weak urinary stream, hesitancy, straining during voiding, intermittency, and the sensation of incomplete bladder emptying [3]. Such manifestations are not only distressing during the day but also interfere with sleep, compounding the patient's discomfort. The underlying mechanisms of BPH are still under investigation, but one widely accepted theory attributes the pathogenesis to hormonal influences, particularly the role of dihydrotestosterone (DHT). DHT is a potent androgen formed by the conversion of testosterone through the action of 5 α -reductase enzymes. Once produced, DHT binds to intracellular androgen receptors within prostatic cells, promoting the transcription of genes that drive cellular proliferation [3]. This results in an increase in prostate volume, which can compress the urethra and lead to the symptomatic presentation characteristic of BPH.

Among the pharmacological treatments available for BPH, finasteride and dutasteride, both classified as 5 α -reductase inhibitors (5-ARIs), have demonstrated efficacy in modifying the disease process. Finasteride inhibits type II

5 α -reductase, thereby reducing DHT levels in the prostate by over 90% and in the serum by up to 70%, regardless of dosage [1][4]. This substantial hormonal suppression leads to a marked decrease in prostate volume, contributing to alleviation of LUTS [2][5][6]. Dutasteride, on the other hand, inhibits both type I and type II isoenzymes, achieving DHT reductions of up to 99% in both the serum and prostate. This broader inhibition enhances its capacity to reduce DHT more effectively across tissues, leading to consistent declines in prostate-specific antigen (PSA) levels—typically by about 50%—and a reduction in prostate volume by approximately 25% over time [3][7]. The reduction in PSA levels caused by 5-ARIs is a critical factor when interpreting laboratory data in men undergoing treatment. Since PSA is used as a biomarker for prostate cancer screening, clinicians must adjust their interpretation in treated patients. A reduction of approximately 50% in PSA concentrations can occur within the first three months of therapy, necessitating a doubling of PSA values for accurate risk assessment in patients who have been on 5-ARIs for six months or longer [8]. Prior to the initiation of 5-ARI therapy, all patients should undergo appropriate prostate cancer screening to establish a baseline and identify any malignancies that may influence treatment planning [8].

The clinical management of BPH includes routine monitoring of symptom severity, often guided by standardized scoring systems such as the American Urological Association (AUA) symptom index. Patients with scores exceeding 10 typically require pharmacologic intervention. The choice of medication depends on multiple factors, including prostate size. Individuals with smaller prostates often respond better to α -adrenergic antagonists, which provide quicker relief of symptoms through smooth muscle relaxation. In contrast, patients with significantly enlarged prostates derive more sustained benefit from 5-ARIs, although the onset of therapeutic effect may take longer, often requiring six to twelve months to reach maximal benefit [3][9]. In practical clinical settings, it is common for BPH management to begin with α -blockers due to their rapid symptomatic relief. When prostate enlargement is substantial or when α -blockers fail to maintain efficacy, 5-ARIs are either introduced concurrently or substituted as monotherapy. This combination approach aligns with evidence supporting the use of dual therapy in patients with moderate-to-severe symptoms and larger prostates. Over time, 5-ARIs contribute to meaningful reductions in prostate size and urinary symptoms, helping to prevent disease progression and reduce the risk of complications such as acute urinary retention and the need for surgical intervention [3][9].

Beyond symptom control and disease modification, 5-ARIs have shown utility in specific perioperative settings. They are often prescribed to patients undergoing transurethral resection of the prostate (TURP) or other prostate surgeries to reduce intraoperative and postoperative bleeding. This effect is thought to be mediated through reductions in prostate vascularity, particularly

microvascular density, although the precise mechanism remains incompletely understood [1][10][11][12][13][14][15]. Several studies have suggested that 5-ARIs lead to histological changes in the prostate, including vascular atrophy and glandular shrinkage, which contribute to lower bleeding risk. To achieve these effects, treatment should ideally begin at least two weeks before the surgical procedure [15]. Research supporting the use of 5-ARIs in this context has demonstrated consistent findings. Dutasteride and finasteride reduce total blood loss and transfusion rates when initiated preoperatively. Histopathological analyses reveal diminished expression of vascular endothelial growth factor (VEGF), a key mediator of angiogenesis, in prostatic tissue exposed to 5-ARIs, further supporting the vascular theory [12][13][14]. By decreasing capillary density and overall blood supply within the prostate, these medications create a surgical field with less bleeding potential, improving operative visibility and reducing complications.

This preoperative benefit is particularly valuable in patients at elevated risk of bleeding or with large prostate volumes. The inclusion of 5-ARIs in perioperative regimens has thus become a recommended practice in urologic guidelines for selected cases. It enhances surgical safety while complementing the long-term management goals of BPH therapy. Additionally, long-term 5-ARI use has been linked to reduced incidence of hematuria in non-surgical patients, especially those with prostate inflammation or prior bleeding episodes [1][10][11]. In summary, finasteride and dutasteride are integral to the pharmacologic management of BPH. They not only relieve urinary symptoms by reducing prostate volume and DHT levels but also offer benefits in terms of PSA modulation and bleeding risk reduction. These effects underscore the importance of patient selection, proper baseline screening, and individualized treatment planning in achieving optimal outcomes. As evidence continues to accumulate, the role of 5-ARIs in both conservative and surgical management of BPH remains firmly established. Their dual utility in long-term symptom control and short-term surgical preparation positions them as essential agents in urologic practice.

Androgenic Alopecia

Androgenic alopecia, also known as male-pattern hair loss, is a widespread dermatological condition that affects both men and women across various age groups. It impacts nearly half of the male population by the age of 40 and appears to affect a comparable proportion of women in the same age category. Additionally, evidence shows that up to 13% of premenopausal women experience varying degrees of this condition [16]. Although androgenic alopecia is not physically debilitating, it causes considerable psychological distress due to the alterations in physical appearance, which can lead to diminished self-esteem and reduced quality of life. Among the various types of alopecia, the androgenic variant accounts for the majority of chronic hair loss cases. The clinical pattern typically begins at the vertex or crown of the scalp and progresses anteriorly, whereas the temporal and occipital regions are often

preserved [16]. The underlying mechanism involves the androgen dihydrotestosterone (DHT), which binds to androgen-sensitive receptors in hair follicles, triggering progressive follicular miniaturization that leads to thinner, shorter, and eventually non-pigmented hair. The physiological response to DHT differs depending on the anatomical location of the hair follicle. In androgen-responsive areas such as the face, DHT induces increased hair growth, which explains the development of facial hair during puberty. In contrast, scalp regions, particularly the crown, exhibit reduced hair density in response to DHT exposure. This dichotomy underscores the hormone's site-specific effects and explains the distinctive pattern of hair loss seen in androgenic alopecia. Although age-related hair thinning is considered a natural part of aging, the condition often begins earlier than expected and significantly impacts younger individuals, leading to heightened emotional vulnerability.

Two pharmacological agents currently approved by the U.S. Food and Drug Administration (FDA) for treating androgenic alopecia are oral finasteride and topical minoxidil. While both agents offer clinical benefit, finasteride is generally regarded as the more effective of the two [17]. These treatments, however, do not provide permanent cures. The therapeutic benefits achieved during active treatment are lost upon cessation, and the hair typically begins to shed within two weeks of stopping finasteride. Within one year of discontinuation, most, if not all, regrown hair is lost, returning the patient to their pre-treatment baseline. Oral finasteride has undergone extensive clinical evaluation to assess its therapeutic efficacy in androgenic alopecia. Numerous controlled studies have confirmed that this agent can significantly reverse hair loss and stimulate regrowth, although results may require up to 12 months to become fully evident. Longitudinal studies have highlighted the contrasting trajectories of hair density between treated and untreated populations. One investigation reported that individuals who did not receive treatment experienced a 26% reduction in hair count over a five-year period. In contrast, those treated with oral finasteride exhibited a 10% increase in hair density within just one year of therapy [2][16][18][19][20][21]. These findings suggest that finasteride not only halts disease progression but may also partially reverse established hair loss.

Additional evidence supporting finasteride's efficacy is provided by clinical trials in which all participants reported increased hair density after 12 months of continuous use [2][6][16][18][19][20][21]. The success rates in clinical practice are also notable. Approximately 88% of men receiving oral finasteride report stabilization of hair loss, and around 66% experience visible regrowth. This is a higher efficacy rate than that observed with topical minoxidil alone, reinforcing finasteride's role as the cornerstone of medical therapy for androgenic alopecia [17]. The positive outcomes associated with finasteride are not confined to monotherapy; when used in conjunction with topical minoxidil, the combination yields superior results

compared to either treatment alone [22][23][24]. Patients treated with both agents benefit from both the vasodilatory effects of minoxidil and the hormonal modulation provided by finasteride. While oral finasteride remains FDA-approved, topical formulations of finasteride, often compounded with minoxidil, have also been evaluated. These combinations have demonstrated improved outcomes compared to monotherapies, but they have not received FDA approval in the United States [23]. In studies comparing the effectiveness of various treatment regimens, the differences in response rates have been significant. One comparative study reported the following improvement rates after 12 months of consistent treatment: 59% of men using 5% topical minoxidil showed noticeable improvement; 80.5% of those taking 1 mg oral finasteride experienced positive results; and 94.1% of patients receiving both treatments demonstrated clinical improvement in hair density and coverage [24]. These figures underscore the additive benefit of combination therapy in managing androgenic alopecia.

The sustained benefit of finasteride therapy also appears to extend beyond the initial year of treatment. Data from five-year follow-up studies show that patients on continuous finasteride maintain or further improve their hair density over time, while untreated individuals continue to experience gradual progression of hair loss [18][19]. These longitudinal observations highlight the importance of ongoing treatment for maintaining clinical gains. The reversible nature of the treatment's effects means that patient education and adherence are critical. Discontinuation not only leads to rapid shedding of newly grown hair but also allows for the resumption of DHT-induced follicular miniaturization. Despite its clinical benefits, the use of finasteride is not without controversy. Some concerns have been raised about potential adverse effects, particularly those involving sexual function, mood, and hormonal balance. Though such side effects are generally infrequent and reversible, they warrant careful discussion with patients prior to initiating therapy. Informed consent and regular monitoring can help mitigate these concerns and support treatment adherence. Physicians must weigh the benefits of hair regrowth against the individual risk profile of the patient, particularly in those with pre-existing psychological or endocrine concerns.

The integration of pharmacotherapy into clinical practice for androgenic alopecia requires a strategic approach. Patients should undergo a comprehensive dermatologic and hormonal evaluation to confirm the diagnosis and rule out other potential causes of hair loss. Once confirmed, treatment planning should consider the patient's age, the severity of hair loss, treatment goals, and psychological impact. Given the chronic nature of the condition and the need for sustained intervention, realistic expectations should be communicated. Although finasteride and minoxidil can significantly improve hair density, neither agent restores hair to pre-alopecia levels, and the degree of response varies among individuals. In sum, androgenic alopecia is a prevalent and distressing condition with

significant psychosocial implications. DHT plays a central role in its pathophysiology, mediating the miniaturization of hair follicles in androgen-sensitive areas of the scalp. Finasteride, by inhibiting the 5 α -reductase enzyme and thereby reducing DHT production, has emerged as one of the most effective pharmacologic treatments for this condition. Long-term use of finasteride stabilizes hair loss in the majority of patients and promotes regrowth in a substantial proportion. The combination of oral finasteride with topical minoxidil further enhances treatment efficacy. However, sustained benefit requires continuous therapy, as cessation of treatment results in a reversal of gains. Clinical decision-making must balance efficacy, side effects, patient preferences, and long-term commitment to therapy. With appropriate patient selection and adherence, finasteride remains a cornerstone of androgenic alopecia management and continues to offer meaningful improvement in both clinical outcomes and patient well-being.

Off-Label Uses

Although topical finasteride has not received FDA approval for the treatment of androgenic alopecia, its use has gained clinical interest due to evidence demonstrating comparable efficacy to the oral formulation. Several studies support that topical finasteride produces similar reductions in scalp dihydrotestosterone (DHT) levels without significantly affecting systemic DHT concentrations [25][26][27]. This localized effect offers a potential therapeutic advantage, particularly for individuals concerned about systemic adverse effects associated with oral finasteride. Despite these findings, the FDA has not officially approved topical finasteride for hair loss treatment, and its use remains off-label. Nonetheless, dermatologists frequently prescribe it as a practical alternative, especially in patients who exhibit intolerance to the oral form.

In contrast, oral minoxidil has an established FDA indication for the treatment of severe hypertension, not hair loss. However, off-label use of oral minoxidil for alopecia is increasingly common due to emerging data supporting its efficacy in stimulating hair growth [28]. Initially formulated as an antihypertensive, oral minoxidil functions as a potent vasodilator, enhancing blood flow to the scalp and promoting follicular stimulation. This mechanism of action mirrors that of topical minoxidil, but the systemic administration allows for more consistent bioavailability. Though not FDA-approved for this purpose, clinicians often recommend low-dose oral minoxidil for patients with extensive or treatment-resistant hair loss. Dutasteride, another 5 α -reductase inhibitor, has not been approved by the FDA for treating hair loss, yet it is widely used off-label for this indication. Like finasteride, dutasteride inhibits the conversion of testosterone to DHT but offers broader enzymatic inhibition by targeting both type I and type II isoforms of 5 α -reductase [29]. This pharmacologic profile results in a more profound and sustained suppression of serum and scalp DHT levels, potentially translating to greater clinical efficacy in managing androgenic alopecia. Comparative studies have suggested that dutasteride may be

as effective as, or even superior to, finasteride in promoting hair regrowth and halting further hair loss [29][30][31][32]. Despite this evidence, dutasteride remains unapproved by the FDA for alopecia management in the United States. Currently, only topical minoxidil and oral finasteride have received official regulatory approval for treating male-pattern baldness [6][16][33][34].

The pharmacokinetic profile of dutasteride, particularly its prolonged half-life, also makes it suitable for localized therapeutic approaches such as scalp microinjection therapy. In this technique, a 0.01% dutasteride solution is administered directly into the scalp, aiming to maximize local drug concentration while minimizing systemic absorption. Although case reports and preliminary studies suggest potential benefit, large-scale randomized controlled trials are currently lacking to confirm its efficacy and safety [21][35][36]. As such, while promising, microinjection therapy with dutasteride remains an experimental modality pending further clinical validation. Beyond dermatologic applications, 5-ARIs have demonstrated potential benefits in oncology and endocrinology. Several studies have reported that these agents may contribute to improved survival outcomes and reduced incidence of bladder cancer [37][38][39][40]. The precise mechanism is not fully established but is believed to involve hormonal modulation and anti-proliferative effects on uroepithelial cells. In patients with prostate cancer, the use of 5-ARIs has been associated with slower disease progression and improved control over tumor growth [41]. These observations have encouraged ongoing investigations into the possible adjunctive use of 5-ARIs in urologic oncology, though their role has not yet been formally integrated into clinical guidelines.

Additionally, 5-ARIs have been employed in the management of idiopathic female hirsutism, although this indication lacks FDA approval. Hirsutism, characterized by excessive hair growth in androgen-sensitive areas, is often treated with hormonal agents that reduce androgen action. Clinical experience has shown that 5-ARIs, by lowering DHT levels, may alleviate symptoms in selected cases of idiopathic hirsutism [42]. While not standard treatment, their off-label use is considered when other therapies fail or are contraindicated. The decision to use 5-ARIs in this population should be guided by careful risk-benefit assessment, given the potential for side effects and the limited scope of supporting evidence. Collectively, these off-label uses illustrate the broader therapeutic potential of 5-ARIs and related agents beyond their FDA-approved indications. Despite the lack of regulatory endorsement for many of these applications, clinical practice often evolves in response to emerging evidence, expert consensus, and patient-specific needs. Nonetheless, the adoption of off-label treatments should be grounded in rigorous clinical judgment, appropriate patient counseling, and awareness of existing safety data. Further high-quality research is necessary to validate these alternative uses and to determine their long-term outcomes and risk profiles.

Mechanism of Action

Androgen synthesis originates in the gonads and, to a lesser extent, the adrenal glands, with cholesterol serving as the initial substrate. The production process results in testosterone, a principal androgen, which circulates in the body and binds to intracellular androgen receptors to mediate its physiological actions. Dihydrotestosterone (DHT), a more potent derivative of testosterone, is formed locally in target tissues through enzymatic conversion by 5 α -reductase isoenzymes, specifically types 1 and 2 [43][44]. Once produced, DHT binds to cytoplasmic androgen receptors, initiating a sequence of intracellular signaling that culminates in the modulation of gene expression within the nucleus.

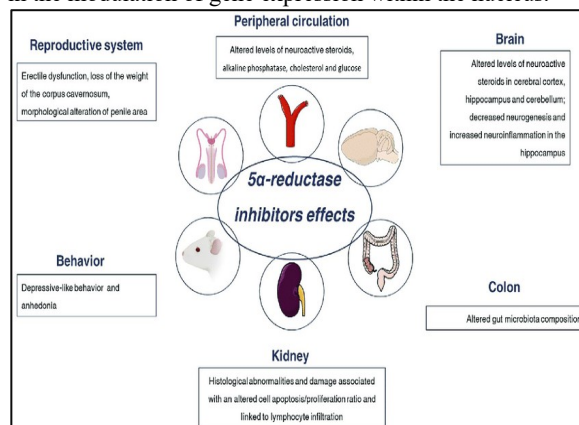


Figure-2: Effects of 5 α -Reductase Inhibitors.

The enzymatic conversion of testosterone to DHT is a critical step in the development of androgen-dependent characteristics. The 5 α -reductase enzymes responsible for this conversion are differentially distributed, with type 1 and type 2 isoforms predominantly located in the scalp and prostate tissues. Although both testosterone and DHT are classified as androgenic steroids, they perform distinct roles. Testosterone is associated with systemic androgenic effects such as increased muscle mass, bone growth, erythropoiesis, and the development of male secondary sexual characteristics during puberty [45]. DHT, on the other hand, plays a more targeted role, particularly in the differentiation of male external genitalia during fetal development, regulation of hair distribution patterns in adulthood, and the proliferation of prostatic cells [43][45][46]. To interfere with the formation of DHT and thereby modulate its biological effects, pharmacologic agents known as 5 α -reductase inhibitors (5-ARIs) are used. Finasteride and dutasteride represent the two main drugs in this category. These agents act as competitive inhibitors of the 5 α -reductase enzyme, thereby preventing the conversion of testosterone to DHT [47]. Finasteride exhibits selective inhibition of the type 2 isoenzyme, whereas dutasteride inhibits both type 1 and type 2 isoforms. This broader inhibition by dutasteride results in a more complete suppression of DHT levels in both the scalp and systemic circulation. Despite this pharmacodynamic distinction, comparative clinical studies have not consistently demonstrated a significant difference in therapeutic outcomes between the two medications for most indications [47].

One of the few clinically meaningful differences between finasteride and dutasteride lies in their pharmacokinetics. Finasteride has a relatively short elimination half-life, ranging between six to eight hours. In contrast, dutasteride has a considerably longer half-life, extending from four to five weeks, which affects both its dosing schedule and drug accumulation in tissues [6][48][49]. This extended duration may contribute to the more sustained suppression of DHT levels observed with dutasteride, although this does not necessarily translate into superior clinical outcomes in all cases. The therapeutic applications of these agents center on conditions that are driven by DHT activity. In benign prostatic hyperplasia (BPH), the reduction in prostatic DHT levels achieved by 5-ARIs results in decreased prostate volume and relief of lower urinary tract symptoms. Similarly, in androgenic alopecia, these agents mitigate follicular miniaturization by reducing DHT activity in the scalp, thereby slowing or reversing hair loss [45]. A recent meta-analysis compared the effectiveness of various therapies for androgenic alopecia and concluded that oral 5-ARIs were more efficacious than both minoxidil and newer approaches such as botulinum toxin injections, microneedling procedures, and photobiomodulation therapies [50]. This reinforces the primary role of 5-ARIs in managing conditions with a strong DHT-mediated pathophysiology.

Administration

Finasteride and dutasteride, the two main 5 α -reductase inhibitors (5-ARIs), are available in distinct dosage forms and strengths tailored to their approved clinical indications. Finasteride is dispensed in oral tablet form in two strengths: 1 mg and 5 mg. The 1 mg formulation is indicated for the treatment of androgenic alopecia, while the 5 mg strength is primarily used in the management of benign prostatic hyperplasia (BPH). Dutasteride is supplied as a 0.5 mg soft gelatin capsule and is prescribed for BPH. For androgenic alopecia, the standard recommended dosage of finasteride is 1 mg per day. For BPH, the dosage increases to 5 mg daily. Dutasteride is typically administered at a dose of 0.5 mg once daily for the treatment of BPH.

Adverse Effects

The adverse effects associated with 5-ARIs primarily involve the reproductive and endocrine systems. The most commonly reported side effects include sexual dysfunction such as erectile dysfunction, reduced libido, and a decrease in ejaculatory volume. Additionally, some patients experience gynecomastia, a condition characterized by the benign enlargement of male breast tissue. These effects are believed to arise from suppressed dihydrotestosterone (DHT) levels and the compensatory increase in aromatization of testosterone to estradiol. Reduced DHT levels disrupt the hormonal balance, potentially leading to both sexual and endocrine changes.

Drug-Drug Interactions

Finasteride and dutasteride function by inhibiting the conversion of testosterone to DHT, thereby increasing circulating testosterone concentrations. A portion of this

excess testosterone may undergo aromatization to estradiol, contributing to gynecomastia in some individuals. Moreover, orthostatic hypotension has been documented, particularly when dutasteride is used in combination with tamsulosin, an α 1-adrenergic receptor antagonist commonly co-prescribed for BPH. This combination can lead to symptomatic hypotension, including dizziness and weakness, which must be carefully monitored, especially in older patients [6].

There are rare but concerning cases in which adverse effects persist even after discontinuation of 5-ARI therapy. This clinical entity, known as post-finasteride syndrome, includes a constellation of symptoms affecting sexual, psychological, and neurological health. Research on this condition is ongoing, and its pathophysiology remains poorly understood. While the majority of patients experience reversal of side effects after drug discontinuation, some report persistent symptoms. Notably, some studies have also observed decreased fertility in men on 5-ARIs. However, this is generally considered reversible upon cessation of therapy, and many men maintain fertility while on treatment [51]. Importantly, α -blockers, often co-administered with 5-ARIs, can also interfere with normal ejaculation by relaxing the smooth muscles of the bladder neck and prostate, further complicating the sexual side effect profile [52][53]. Post-finasteride syndrome is increasingly recognized in clinical literature and is marked by symptoms that continue beyond the discontinuation of either finasteride or dutasteride. Nearly all affected patients report persistent sexual dysfunction, but mental health concerns are also prevalent. Cases have included reports of anxiety, depression, and even suicidal ideation following cessation of 5-ARI therapy [54][55][56][57][58]. The lack of an effective treatment further complicates management, and patients experiencing such symptoms should be referred for multidisciplinary care, including urology, psychiatry, and endocrinology evaluation.

Contraindications

Use of 5-ARIs is contraindicated in certain populations due to the hormonal effects of these agents. As DHT plays a pivotal role in male sexual development, the administration of 5-ARIs in children and pregnant women is contraindicated. Particularly, the drugs should not be handled by women who are or may become pregnant due to the potential teratogenic risk, specifically the risk of disrupting fetal male genital development [59]. Additionally, individuals with known hypersensitivity to finasteride or dutasteride should avoid their use, and alternative therapies should be considered [6].

Monitoring

Although formal guidelines for monitoring the effects of 5-ARI therapy are not universally established, prostate-specific antigen (PSA) levels are frequently used to assess therapeutic response in patients with BPH. Since the prostate is the primary source of PSA production, reductions in prostate size brought on by 5-ARIs correspond with lowered PSA levels. Thus, serial measurement of PSA can

serve as an indirect indicator of drug efficacy in BPH management [6][60]. However, clinicians must recognize that 5-ARIs significantly suppress PSA levels, often by approximately 50% after six months of therapy. For accurate interpretation, measured PSA values in patients on long-term 5-ARI therapy should be doubled when considering cancer risk or screening outcomes [3][7]. The relationship between finasteride use and prostate cancer risk has been controversial. The Prostate Cancer Prevention Trial (PCPT) evaluated the long-term impact of finasteride on cancer incidence among men aged 55 and older. This study reported a 25% reduction in overall prostate cancer prevalence among those taking finasteride. However, it also identified a higher incidence of high-grade tumors in the treatment group, leading to significant concern [61]. Based on these results, the U.S. Food and Drug Administration (FDA) issued a boxed warning on finasteride to highlight this potential risk [62]. Yet, follow-up investigations and independent analyses have challenged these findings. Subsequent studies have failed to confirm an increased incidence of aggressive prostate cancer associated with 5-ARI use and have instead suggested the possibility of improved detection accuracy due to reduced prostate volume [63][64][65][66]. Given these complexities, PSA monitoring remains an essential aspect of long-term management in men taking 5-ARIs, particularly those with elevated baseline PSA or a family history of prostate cancer. Regular follow-up with urologic evaluation is advisable for men on long-term therapy, and any rapid rise in PSA, even from a reduced baseline, should prompt further diagnostic assessment.

Toxicity

To date, documented reports of acute toxicity from either finasteride or dutasteride are lacking. These agents are generally well tolerated and do not exhibit toxicity profiles commonly associated with other pharmacological classes. Overdose cases are rare, and no specific antidotes exist, as supportive care is typically sufficient to manage adverse events. Routine use in clinical settings supports their safety when administered within the recommended dosage range.

Enhancing Healthcare Team Outcomes

The clinical effectiveness of 5-ARIs in treating both BPH and androgenic alopecia is well established. However, optimal outcomes require the coordinated efforts of a multidisciplinary healthcare team. Physicians play a central role in diagnosis, patient selection, and therapeutic planning. Nurses and pharmacists are essential in patient education, ensuring adherence to the prescribed regimen, and monitoring adverse effects. Given the chronic nature of BPH and androgenic alopecia, long-term compliance is essential for sustained benefit. Clear communication about the onset of therapeutic effects, which may take several months, helps align patient expectations and prevents premature discontinuation. Screening for prostate cancer prior to initiating therapy is critical, especially in men over 50 or with risk factors for malignancy. Baseline PSA testing should be performed, and any abnormalities should be fully evaluated before beginning 5-ARI treatment. Patients must also be counseled on potential side effects, including those

affecting sexual function, and advised that some symptoms may persist even after stopping treatment. Education on the reversibility of most adverse effects can help reduce anxiety and improve adherence. Finally, the role of 5-ARIs in healthcare extends beyond symptomatic relief. Their potential to reduce surgical interventions in BPH, their contribution to stabilizing progressive hair loss, and their emerging roles in cancer prevention all indicate their broader clinical value. As with any long-term therapy, individualized care, regular monitoring, and proactive management of adverse events are key to maximizing the therapeutic benefit of 5-ARIs and ensuring favorable patient outcomes.

Conclusion:

5 α -reductase inhibitors (5-ARIs) have revolutionized the management of androgen-dependent conditions, particularly BPH and androgenic alopecia. By inhibiting DHT production, finasteride and dutasteride alleviate lower urinary tract symptoms, reduce prostate volume, and slow hair loss progression. Clinical evidence underscores their efficacy: finasteride increases hair density in 66% of alopecia patients, while dutasteride achieves near-complete DHT suppression. However, their benefits are tempered by adverse effects, including sexual dysfunction and gynecomastia, which may persist as post-finasteride syndrome in rare cases. The perioperative utility of 5-ARIs in reducing BPH-related bleeding further highlights their versatility. Off-label applications, such as dutasteride microinjections for alopecia and chemoprevention in prostate cancer, demonstrate expanding therapeutic potential but lack robust FDA endorsement. Monitoring PSA levels in BPH patients is critical, as 5-ARIs halve PSA values, necessitating adjusted interpretations to avoid masking prostate cancer. A collaborative healthcare approach—integrating urologists, dermatologists, pharmacists, and primary care providers—is essential for optimizing 5-ARI therapy. Patient education on delayed onset of effects (6–12 months) and adherence is vital to prevent discontinuation. While 5-ARIs are generally safe, contraindications (e.g., pregnancy, pediatric use) and drug interactions (e.g., with α -blockers) warrant vigilance. Future research should address persistent knowledge gaps, including the pathophysiology of post-finasteride syndrome and long-term outcomes of off-label uses. For now, 5-ARIs remain indispensable in androgen-mediated disorders, provided their use is guided by individualized risk assessment and multidisciplinary oversight to balance efficacy with tolerability.

References:

1. Smith AB, Carson CC. Finasteride in the treatment of patients with benign prostatic hyperplasia: a review. *Therapeutics and clinical risk management*. 2009 Jun;5(3):535-45
2. Shin JW, Chung EH, Kim MB, Kim TO, Kim WI, Huh CH. Evaluation of long-term efficacy of finasteride in Korean men with androgenetic alopecia using the basic and specific classification system. *The Journal of dermatology*. 2019 Feb;46(2):139-143. doi: 10.1111/1346-8138.14719.

3. Ng M, Leslie SW, Baradhi KM. Benign Prostatic Hyperplasia. StatPearls. 2024 Jan
4. Norman RW, Coakes KE, Wright AS, Rittmaster RS. Androgen metabolism in men receiving finasteride before prostatectomy. The Journal of urology. 1993 Nov;150(5 Pt 2):1736-9
5. Treatments for Benign Prostatic Hyperplasia. 2004 Aug 2
6. Zito PM, Bistas KG, Patel P, Syed K. Finasteride. StatPearls. 2024 Jan
7. Hernandez J, Gelfond J, Goros M, Liss MA, Liang Y, Ankerst D, Thompson IM Jr, Leach RJ. The effect of 3-month finasteride challenge on biomarkers for predicting cancer outcome on biopsy: Results of a randomized trial. PloS one. 2018;13(10):e0204823. doi: 10.1371/journal.pone.0204823.
8. Jain MA, Leslie SW, Sapra A. Prostate Cancer Screening. StatPearls. 2024 Jan
9. Nachawati D, Patel JB. Alpha-Blockers. StatPearls. 2024 Jan
10. Puchner PJ, Miller MI. The effects of finasteride on hematuria associated with benign prostatic hyperplasia: a preliminary report. The Journal of urology. 1995 Nov;154(5):1779-82
11. Miller MI, Puchner PJ. Effects of finasteride on hematuria associated with benign prostatic hyperplasia: long-term follow-up. Urology. 1998 Feb;51(2):237-40
12. Foley SJ, Solomon LZ, Wedderburn AW, Kashif KM, Summerton D, Basketter V, Holmes SA. A prospective study of the natural history of hematuria associated with benign prostatic hyperplasia and the effect of finasteride. The Journal of urology. 2000 Feb;163(2):496-8
13. Donohue JF, Hayne D, Karnik U, Thomas DR, Foster MC. Randomized, placebo-controlled trial showing that finasteride reduces prostatic vascularity rapidly within 2 weeks. BJU international. 2005 Dec;96(9):1319-22
14. Memis A, Ozden C, Ozdal OL, Guzel O, Han O, Seckin S. Effect of finasteride treatment on suburethral prostatic microvessel density in patients with hematuria related to benign prostate hyperplasia. Urologia internationalis. 2008;80(2):177-80. doi: 10.1159/000112610.
15. Ozdal OL, Ozden C, Benli K, Gökkaya S, Bulut S, Memiş A. Effect of short-term finasteride therapy on peroperative bleeding in patients who were candidates for transurethral resection of the prostate (TUR-P): a randomized controlled study. Prostate cancer and prostatic diseases. 2005;8(3):215-8
16. Ho CH, Sood T, Zito PM. Androgenetic Alopecia. StatPearls. 2024 Jan
17. Arca E, Açıkgöz G, Taştan HB, Köse O, Kurumlu Z. An open, randomized, comparative study of oral finasteride and 5% topical minoxidil in male androgenetic alopecia. Dermatology (Basel, Switzerland). 2004;209(2):117-25
18. Sasaki GH. Review of Human Hair Follicle Biology: Dynamics of Niches and Stem Cell Regulation for Possible Therapeutic Hair Stimulation for Plastic Surgeons. Aesthetic plastic surgery. 2019 Feb;43(1):253-266. doi: 10.1007/s00266-018-1248-1.
19. Feingold KR, Anawalt B, Blackman MR, Boyce A, Chrousos G, Corpas E, de Herder WW, Dhatariya K, Dungan K, Hofland J, Kalra S, Kaltsas G, Kapoor N, Koch C, Kopp P, Korbonits M, Kovacs CS, Kuohung W, Laferrère B, Levy M, McGee EA, McLachlan R, New M, Purnell J, Sahay R, Shah AS, Singer F, Sperling MA, Stratakis CA, Trencle DL, Wilson DP, Asfour L, Cranwell W, Sinclair R. Male Androgenetic Alopecia. Endotext. 2000
20. Stough DB, Rao NA, Kaufman KD, Mitchell C. Finasteride improves male pattern hair loss in a randomized study in identical twins. European journal of dermatology : EJD. 2002 Jan-Feb;12(1):32-7
21. Courtney A, Triwongwarant D, Chim I, Eisman S, Sinclair R. Evaluating 5 alpha reductase inhibitors for the treatment of male androgenic alopecia. Expert opinion on pharmacotherapy. 2023 Sep-Dec;24(18):1919-1922. doi: 10.1080/14656566.2023.2280630.
22. Chen L, Zhang J, Wang L, Wang H, Chen B. The Efficacy and Safety of Finasteride Combined with Topical Minoxidil for Androgenetic Alopecia: A Systematic Review and Meta-analysis. Aesthetic plastic surgery. 2020 Jun;44(3):962-970. doi: 10.1007/s00266-020-01621-5.
23. Rossi A, Caro G. Efficacy of the association of topical minoxidil and topical finasteride compared to their use in monotherapy in men with androgenetic alopecia: A prospective, randomized, controlled, assessor blinded, 3-arm, pilot trial. Journal of cosmetic dermatology. 2024 Feb;23(2):502-509. doi: 10.1111/jocd.15953.
24. Hu R, Xu F, Sheng Y, Qi S, Han Y, Miao Y, Rui W, Yang Q. Combined treatment with oral finasteride and topical minoxidil in male androgenetic alopecia: a randomized and comparative study in Chinese patients. Dermatologic therapy. 2015 Sep-Oct;28(5):303-8. doi: 10.1111/dth.12246.
25. Abeck F, Hansen I, Kött J, Schröder F, Garrahy E, Veneroso J, Rünger A, Torster L, Schneider SW, von Büren J. Patient-reported outcomes of topical finasteride/minoxidil treatment for male androgenetic alopecia: A retrospective study using telemedical data. Journal of cosmetic dermatology. 2024 May 7(0):. doi: 10.1111/jocd.16360.

26. Keerti A, Madke B, Keerti A, Lopez MJC, Lirio FS. Topical Finasteride: A Comprehensive Review of Androgenetic Alopecia Management for Men and Women. *Cureus*. 2023 Sep;15(9):e44949. doi: 10.7759/cureus.44949.
27. Piraccini BM, Blume-Peytavi U, Scarci F, Jansat JM, Falqués M, Otero R, Tamarit ML, Galván J, Tebbs V, Massana E, Topical Finasteride Study Group. Efficacy and safety of topical finasteride spray solution for male androgenetic alopecia: a phase III, randomized, controlled clinical trial. *Journal of the European Academy of Dermatology and Venereology : JEADV*. 2022 Feb;36(2):286-294. doi: 10.1111/jdv.17738.
28. Gupta AK, Talukder M, Shemer A. Efficacy and safety of low-dose oral minoxidil in the management of androgenetic alopecia. *Expert opinion on pharmacotherapy*. 2024 Feb;25(2):139-147. doi: 10.1080/14656566.2024.2314087.
29. Arif T, Dorjay K, Adil M, Sami M. Dutasteride in Androgenetic Alopecia: An Update. *Current clinical pharmacology*. 2017;12(1):31-35. doi: 10.2174/1574884712666170310111125.
30. Dhurat R, Sharma A, Rudnicka L, Kroumpouzos G, Kassir M, Galadari H, Wollina U, Lotti T, Golubovic M, Binic I, Grabbe S, Goldust M. 5-Alpha reductase inhibitors in androgenetic alopecia: Shifting paradigms, current concepts, comparative efficacy, and safety. *Dermatologic therapy*. 2020 May;33(3):e13379. doi: 10.1111/dth.13379.
31. Choi S, Kwon SH, Sim WY, Lew BL. Long-term efficacy and safety of dutasteride 0.5 mg in Korean men with androgenetic alopecia: 5-year data demonstrating clinical improvement with sustained efficacy. *The Journal of dermatology*. 2024 May;51(5):684-690. doi: 10.1111/1346-8138.17138.
32. Gupta AK, Talukder M, Williams G. Comparison of oral minoxidil, finasteride, and dutasteride for treating androgenetic alopecia. *The Journal of dermatological treatment*. 2022 Nov;33(7):2946-2962. doi: 10.1080/09546634.2022.2109567.
33. Gupta AK, Talukder M, Williams G. Emerging and traditional 5- α reductase inhibitors and androgen receptor antagonists for male androgenetic alopecia. *Expert opinion on emerging drugs*. 2024 Apr 26;():1-11. doi: 10.1080/14728214.2024.2346590.
34. Patel P, Nessel TA, Kumar D D. Minoxidil. *StatPearls*. 2024 Jan
35. Saceda-Corralo D, Moustafta F, Moreno-Arrones Ó, Jaén-Olasolo P, Vaño-Galván S, Camacho F. Mesotherapy With Dutasteride for Androgenetic Alopecia: A Retrospective Study in Real Clinical Practice. *Journal of drugs in dermatology : JDD*. 2022 Jul 1:21(7):742-747. doi: 10.36849/JDD.6610.
36. Rodríguez-Cuadrado FJ, Pinto-Pulido EL, Fernández-Parrado M. Mesotherapy with dutasteride for androgenetic alopecia: a concise review of the literature. *European journal of dermatology : EJD*. 2023 Feb 1:33(1):72. doi: 10.1684/ejd.2023.4443.
37. Wang Y, Song Y, Peng Y, Han S, Qin C, Du Y, Xu T. Effects of androgen suppression therapy on the incidence and prognosis of bladder cancer: An updated systematic review and meta-analysis. *Urologic oncology*. 2024 May 9;(): pii: S1078-1439(24)00431-9. doi: 10.1016/j.urolonc.2024.04.014.
38. Pyun JH, Son NH, Ko YH, Kim SW, Kim H, Bae YJ. The Long-Term Impact of 5-alpha Reductase Inhibitors on the Development of Bladder Cancer and the Need for Radical Cystectomy: A Nationwide Observational Study. *The world journal of men's health*. 2024 Apr;42(2):460-466. doi: 10.5534/wjmh.230137.
39. An MH, Kim MS, Kim C, Noh TI, Joo KJ, Lee DH, Yi KH, Kwak JW, Hwang TH, Park RW, Kang SH. Association of 5 α -Reductase Inhibitor Prescription With Bladder Cancer Progression in Males in South Korea. *JAMA network open*. 2023 May 1:6(5):e2313667. doi: 10.1001/jamanetworkopen.2023.13667
40. Garg H, Wheeler KM, Dursun F, Cooper RE, Pruthi DK, Kaushik D, Thompson IM, Svatek RS, Liss MA. Impact of Finasteride on Survival in Bladder Cancer: A Retrospective Multi-institutional Database Analysis. *Clinical genitourinary cancer*. 2023 Apr;21(2):314.e1-314.e7. doi: 10.1016/j.clgc.2022.10.014.
41. Matsukawa A, Yanagisawa T, Bekku K, Parizi MK, Laukhtina E, Klemm J, Chiujdea S, Mori K, Kimura S, Miki J, Pradere B, Rivas JG, Gandaglia G, Kimura T, Kasivisvanathan V, Ploussard G, Cornford P, Shariat SF, Rajwa P. Nonsurgical Interventions to Prevent Disease Progression in Prostate Cancer Patients on Active Surveillance: A Systematic Review and Meta-analysis. *European urology oncology*. 2024 Jun;7(3):376-400. doi: 10.1016/j.euo.2023.10.010.
42. Moghetti P, Castello R, Magnani CM, Tosi F, Negri C, Armanini D, Bellotti G, Muggeo M. Clinical and hormonal effects of the 5 alpha-reductase inhibitor finasteride in idiopathic hirsutism. *The Journal of clinical endocrinology and metabolism*. 1994 Oct;79(4):1115-21
43. Kinter KJ, Amraei R, Anekar AA. Biochemistry, Dihydrotestosterone. *StatPearls*. 2025 Jan
44. Marchetti PM, Barth JH. Clinical biochemistry of dihydrotestosterone. *Annals of clinical*

- biochemistry. 2013 Mar;50(Pt 2):95-107. doi: 10.1258/acb.2012.012159.
45. Nassar GN, Leslie SW. Physiology, Testosterone. StatPearls. 2024 Jan(:):
46. Basaria S. Reproductive aging in men. Endocrinology and metabolism clinics of North America. 2013 Jun;42(2):255-70. doi: 10.1016/j.ecl.2013.02.012.
47. Nickel JC. Comparison of clinical trials with finasteride and dutasteride. Reviews in urology. 2004;6 Suppl 9(Suppl 9):S31-9
48. Erdemir F, Harbin A, Hellstrom WJ. 5-alpha reductase inhibitors and erectile dysfunction: the connection. The journal of sexual medicine. 2008 Dec;5(12):2917-24. doi: 10.1111/j.1743-6109.2008.01001.x.
49. Gisleskog PO, Hermann D, Hammarlund-Udenaes M, Karlsson MO. The pharmacokinetic modelling of GI198745 (dutasteride), a compound with parallel linear and nonlinear elimination. British journal of clinical pharmacology. 1999 Jan;47(1):53-8
50. Gupta AK, Bamimore MA, Wang T, Talukder M. The impact of monotherapies for male androgenetic alopecia: A network meta-analysis study. Journal of cosmetic dermatology. 2024 May 9(:):. doi: 10.1111/jocd.16362.
51. Andrade C. Why Odds Ratios Can Be Tricky Statistics: The Case of Finasteride, Dutasteride, and Sexual Dysfunction. The Journal of clinical psychiatry. 2018 Nov 27;79(6):. pii: 18f12641. doi: 10.4088/JCP.18f12641.
52. Bearely P, Avellino GJ. The role of benign prostatic hyperplasia treatments in ejaculatory dysfunction. Fertility and sterility. 2021 Sep;116(3):611-617. doi: 10.1016/j.fertnstert.2021.07.1199.
53. Bapir R, Bhatti KH, Eliwa A, García-Perdomo HA, Gherabi N, Hennessey D, Magri V, Mourmouris P, Ouattara A, Perletti G, Philipraj J, Trinchieri A, Buchholz N. Effect of alpha-adrenoceptor antagonists on sexual function. A systematic review and meta-analysis. Archivio italiano di urologia, andrologia : organo ufficiale [di] Societa italiana di ecografia urologica e nefrologica. 2022 Jun 30;94(2):252-263. doi: 10.4081/aiua.2022.2.252.
54. Traish AM. Post-finasteride syndrome: a surmountable challenge for clinicians. Fertility and sterility. 2020 Jan;113(1):21-50. doi: 10.1016/j.fertnstert.2019.11.030.
55. Coskuner ER, Ozkan B, Culha MG. Sexual Problems of Men With Androgenic Alopecia Treated With 5-Alpha Reductase Inhibitors. Sexual medicine reviews. 2019 Apr;7(2):277-282. doi: 10.1016/j.sxmr.2018.07.003.
56. Said MA, Mehta A. The Impact of 5 α -Reductase Inhibitor Use for Male Pattern Hair Loss on Men's Health. Current urology reports. 2018 Jun 16;19(8):65. doi: 10.1007/s11934-018-0814-z.
57. Fertig RM, Gamret AC, Darwin E, Gaudi S. Sexual side effects of 5- α -reductase inhibitors finasteride and dutasteride: A comprehensive review. Dermatology online journal. 2017 Nov 11;23(11):. pii: 13030/qt24k8q743.
58. Lee S, Lee YB, Choe SJ, Lee WS. Adverse Sexual Effects of Treatment with Finasteride or Dutasteride for Male Androgenetic Alopecia: A Systematic Review and Meta-analysis. Acta dermato-venereologica. 2019 Jan 1;99(1):12-17. doi: 10.2340/00015555-3035.
59. Teichert M, van Puijenbroek E, Stricker BH. Contraindicated use of 5-alpha-reductase inhibitors in women. British journal of clinical pharmacology. 2017 Feb;83(2):429-431. doi: 10.1111/bcp.13118.
60. Andy G, John M, Mirna S, Rachita D, Michael K, Maja K, Aseem S, Zeljana B. Controversies in the treatment of androgenetic alopecia: The history of finasteride. Dermatologic therapy. 2019 Mar;32(2):e12647. doi: 10.1111/dth.12647.
61. Goodman PJ, Tangen CM, Darke AK, Lucia MS, Ford LG, Minasian LM, Parnes HL, LeBlanc ML, Thompson IM Jr. Long-Term Effects of Finasteride on Prostate Cancer Mortality. The New England journal of medicine. 2019 Jan 24;380(4):393-394. doi: 10.1056/NEJMc1809961.
62. Shin YS, Karna KK, Choi BR, Park JK. Finasteride and Erectile Dysfunction in Patients with Benign Prostatic Hyperplasia or Male Androgenetic Alopecia. The world journal of men's health. 2019 May;37(2):157-165. doi: 10.5534/wjmh.180029.
63. Hirshburg JM, Kelsey PA, Therrien CA, Gavino AC, Reichenberg JS. Adverse Effects and Safety of 5-alpha Reductase Inhibitors (Finasteride, Dutasteride): A Systematic Review. The Journal of clinical and aesthetic dermatology. 2016 Jul;9(7):56-62
64. Bonde Miranda T, Garmo H, Stattin P, Robinson D. 5 α -Reductase Inhibitors and Risk of Prostate Cancer Death. The Journal of urology. 2020 Oct;204(4):714-719. doi: 10.1097/JU.0000000000001038
65. Vaselkiv JB, Ceraolo C, Wilson KM, Pernar CH, Rencsok EM, Stopsack KH, Grob ST, Plym A, Giovannucci EL, Olumi AF, Kibel AS, Preston MA, Mucci LA. 5-Alpha Reductase Inhibitors and Prostate Cancer Mortality among Men with Regular Access to Screening and Health Care. Cancer epidemiology, biomarkers & prevention : a publication of the American Association for

-
- Cancer Research, cosponsored by the American Society of Preventive Oncology. 2022 Jul 1;31(7):1460-1465. doi: 10.1158/1055-9965.EPI-21-1234.
66. Unger JM, Till C, Thompson IM Jr, Tangen CM, Goodman PJ, Wright JD, Barlow WE, Ramsey SD, Minasian LM, Hershman DL. Long-term Consequences of Finasteride vs Placebo in the Prostate Cancer Prevention Trial. Journal of the National Cancer Institute. 2016 Dec;108(12):. pii: djw168. doi: 10.1093/jnci/djw168. Epub 2016 Aug 26