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# Adverse effects of finasteride in men treated for benign prostatic hyperplasia and androgenetic alopecia - a literature review

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**Abstract**

**Background** Finasteride, a selective inhibitor of the enzyme 5-alpha reductase type II, is extensively utilized in the management of androgenetic alopecia (AGA) and benign prostatic hyperplasia (BPH). By reducing the conversion of testosterone to dihydrotestosterone (DHT), finasteride effectively mitigates the pathophysiological mechanisms underlying these conditions. Despite its proven efficacy, the therapeutic use of finasteride has been associated with a spectrum of adverse effects, particularly in the domains of sexual, neuropsychiatric, and endocrine health. This review aims to comprehensively analyze the incidence, pathophysiology, and clinical management of adverse effects related to finasteride therapy.

**Material and methods** A comprehensive search of databases, including PubMed and Google Scholar, was conducted using the keywords mentioned below.

**Conclusions** Finasteride is considered a safe and effective medication for the treatment of androgenetic alopecia and benign prostatic hyperplasia, with a well-documented safety profile. However, its use may be associated with adverse effects, including sexual dysfunction and psychological symptoms, which can impact patient satisfaction and adherence to treatment. Although these side effects are typically reversible upon discontinuation, clinicians should discuss potential risks with patients and implement monitoring strategies to ensure optimal outcomes and patient confidence in therapy.

**Keywords:** finasteride, 5α-reductase inhibitor, androgenetic alopecia, benign prostatic hyperplasia**.**

## Introduction

Finasteride is a 5α-reductase inhibitor (5ARI) that binds irreversibly to the type II 5α-reductase enzyme, inhibiting the conversion of testosterone to dihydrotestosterone (DHT). It also inhibits the type III 5α-reductase isoenzyme, albeit to a lesser extent. Since 1992, finasteride has been widely used in the treatment of benign prostatic hyperplasia (BPH) at a dose of 5 mg. Five years later, in 1997, it was approved for the treatment of androgenetic alopecia at a lower dose of 1 mg. Due to its efficacy and well-established use, it remains a popular choice among both physicians and patients. Finasteride is widely regarded as safe and effective when used as prescribed. Reported adverse effects include a reduction in libido, erectile dysfunction, and ejaculatory dysfunction. Less common side effects include gynecomastia, depression, anxiety, and suicidal ideation. Studies have shown that the prevalence of sexual dysfunction does not significantly increase when the dosage is raised from 1 mg to 5 mg daily [1]. Additionally, long-term use of finasteride 5 mg daily in men over 55 years has been shown to reduce the risk of low-grade prostate cancer by approximately 25%. However, some studies have reported an increased detection rate of high-grade prostate cancer among those diagnosed while using finasteride, potentially due to improved diagnostic accuracy related to reduced prostate volume. Whether these benefits or risks occur in men under 55 years taking 1 mg daily is not yet established [2]. Furthermore, finasteride 5 mg may mildly affect semen parameters, but these changes typically resolve within weeks of discontinuation, particularly in men with fertility concerns [3]. Following an FDA warning in 2012 about persistent sexual and neuropsychiatric effects, reports of these adverse events increased. Most adverse effects associated with finasteride resolve within 3 to 6 months after discontinuing the medication [4]. However, a small percentage of users may persist for a longer duration, even after stopping the treatment. These prolonged adverse effects, while uncommon, underscore the importance of careful monitoring and patient education regarding the potential risks of finasteride use [5].

## Sexual Dysfunction

Sexual dysfunction is one of the most consistently reported adverse effects of finasteride use, with both observational studies and numerous clinical trials establishing an association between finasteride intake and complications such as erectile dysfunction, decreased libido, and ejaculatory dysfunction. Persistent sexual dysfunction (PSD) refers to sexual dysfunction that lasts for at least 90 days after discontinuing the causative drug. This time frame distinguishes PSD from transient sexual side effects that typically resolve shortly after stopping treatment.

A retrospective cohort study of 1,390 men treated with finasteride 1 mg and a control group of 20,000 omeprazole users found that the rate of PSD was 37.9 per 1,000 person-years for finasteride users, significantly higher than 15 per 1,000 person-years in the control group. The adjusted hazard ratio (HR) for PSD was 1.62 (95% CI: 1.14–2.29). When PSD was defined as requiring phosphodiesterase inhibitors such as sildenafil, the adjusted HR rose to 2.73 (95% CI: 2.01–3.69). Furthermore, the median time to the first PSD event after stopping finasteride was 339 days, with some cases extending up to 1,680 days [6].

**Erectile Dysfunction**

The use of finasteride, a 5-alpha reductase inhibitor, has been associated with an increased risk of erectile dysfunction (ED) in men treated for androgenetic alopecia (AGA) or benign prostatic hyperplasia (BPH). The mechanism underlying this side effect is thought to involve the suppression of dihydrotestosterone (DHT), a key androgen responsible for the maintenance of penile tissue and nitric oxide synthase activity in the corpus cavernosum. Reduced nitric oxide levels may impair penile vasodilation, which is crucial for normal erectile function [7] Numerous studies have assessed the prevalence of ED among finasteride users, reporting rates ranging from 0.8% to 15.8%, depending on dosage and study population [1,7,8]. A meta-analysis of 12 randomized trials involving 3,927 participants found a relative risk (RR) of ED of 2.22 (95% CI, 1.03–4.78) compared to placebo, although most cases resolved upon discontinuation [8]. However, persistent erectile dysfunction (PED)—defined as ED lasting more than 90 days after cessation of the drug—has been observed. A study of 11,909 men revealed a PED incidence of 1.4%, with a median duration of 1,348 days post-finasteride use . Younger men, especially those aged 16–42 years and exposed to the drug for longer than 205 days, exhibited a nearly 4.9-fold increased risk of PED [9]. Long-term studies provide mixed conclusions about the risk of ED with finasteride. For example, the Prostate Cancer Prevention Trial (PCPT), involving 17,313 participants, found a slight increase in sexual dysfunction scores among finasteride users compared to placebo; however, the impact diminished over time and was minimal at the 7-year follow-up [4]. Additionally, a survey-based study using the Arizona Sexual Experience Scale (ASEX) found no significant difference in ED prevalence between finasteride users and non-users [10].
Conversely, more recent studies have documented persistent sexual side effects in a subset of men. One such study involving 71 participants reported new-onset persistent ED and decreased libido lasting an average of 40 months after stopping the drug. However, this study faced limitations such as small sample size, recall bias, and a lack of serum hormone analyses, making the findings less generalizable . The psychological dimension, including the nocebo effect, plays a role in the reported ED cases. A study demonstrated that patients informed about potential sexual side effects experienced higher rates of ED compared to uninformed patients. These symptoms resolved within five days of discontinuation, underscoring the influence of psychological factors[6]

**Decreased Libido**

In clinical trials, the incidence of decreased libido in patients taking finasteride has been shown to be dose-dependent. For example, a study involving 895 participants treated with 5 mg of finasteride daily for one year reported that 4.7% of patients experienced a decrease in libido, compared to 1.3% in the placebo group [11]. Similarly, in a study involving 326 men treated with 1 mg/day finasteride, 2% reported decreased libido. These findings suggest that sexual side effects, including libido loss, are more common in the finasteride group compared to those receiving a placebo [12]. Further observational studies confirm these findings. A cohort study of 14,772 men treated with 5 mg of finasteride for BPH found that 1% of patients reported a decrease in libido. The mechanism behind finasteride's impact on libido is related to its inhibition of 5-alpha-reductase. DHT is a potent androgen that plays a critical role in male sexual function. By reducing DHT levels, finasteride may impair the signaling pathways involved in sexual desire and arousal, which can result in a decrease in libido. This hormonal imbalance is thought to contribute to the sexual side effects seen in finasteride users [13].

**Ejaculatory Dysfunction**

The reduction of DHT leads to decreased prostatic fluid production, which constitutes a significant component of seminal fluid. This mechanism could explain the observed reduction in ejaculate volume and the occurrence of delayed ejaculation among finasteride users .
The Finasteride Male Pattern Hair Loss Study observed that reduced ejaculatory volume was slightly more prevalent in participants receiving finasteride compared to those on placebo. In the first year, the incidence of reduced ejaculatory volume in the finasteride group was 1.0%, compared to 0.4% in the placebo group during the extension phase of the study [14]. Another study involved a long-term observation of patients with benign prostatic hyperplasia (BPH) who were treated with finasteride at a dose of 5 mg daily for 7 to 8 years.The study

included 190 men, of whom 156 continued therapy during the open-label phase, and 71 completed the full 7 years of treatment. The analysis focused on drug-related sexual adverse effects, including ejaculatory dysfunction. The incidence of ejaculatory dysfunction decreased over time, starting at 5.8% in the first year, 0.8% in the second year, 1.0% in the third year, and stabilizing at 1.1% annually during years four to seven [15] . Mechanistically, the reduction in DHT levels also affects the neuromuscular pathways involved in ejaculation, potentially contributing to delayed ejaculation or incomplete ejaculation. This hormonal imbalance may explain the broader spectrum of ejaculatory dysfunction observed in finasteride users [16].

**Impact on spermatogenesis**

The reduction in DHT levels may interfere with Sertoli cell function and impair spermatogenesis. Additionally, some studies have linked finasteride to increased sperm DNA fragmentation, which could contribute to fertility issues. However, this finding is not universally consistent across all studies, suggesting that the full extent of finasteride's effect on sperm quality is still not entirely understood . In one randomized controlled trial involving 1 mg/day of finasteride administered to healthy men, semen parameters, including sperm concentration, motility, and morphology, were not significantly affected after six months of treatment. However, a mild reduction in semen volume was noted, which normalized within three months of discontinuation. This suggests that finasteride-induced effects are reversible in most cases [17]. A notable case study involving a 42-year-old man with azoospermia reported rapid improvement in sperm concentration to over $10 \times 10^6$/mL within 16 weeks of stopping finasteride. Similarly, a retrospective analysis indicated a significant increase in sperm count following discontinuation in men with severe oligospermia, emphasizing the potential for reversibility [18]. The impact appears to be dose-dependent. Men taking the 5 mg dose of finasteride for benign prostatic hyperplasia (BPH) demonstrated more pronounced reductions in sperm count, motility, and semen volume compared to those on the 1 mg dose used for androgenetic alopecia. These changes were reversible but were observed more frequently in men with pre-existing fertility issues . It should be mentioned that studies on healthy men generally do not report significant fertility issues related to finasteride. For instance, in a cohort study of men on 1 mg/day, there were no statistically significant differences in conception rates compared to controls. However, the findings suggest caution when prescribing finasteride to men with subfertility or those planning to conceive. The reversibility of spermatogenic changes has been documented across multiple studies, with recovery typically occurring within 3–6 months of stopping finasteride. However, in rare cases, persistent abnormalities in sperm parameters have been reported, especially in men on long-term therapy or with pre-existing reproductive issues [17,19].

**Depression**

Several studies have identified a link between finasteride use and adverse psychological outcomes, including depression. Finasteride inhibits the 5-alpha reductase enzyme, which reduces the production of neurosteroids like allopregnanolone. These neurosteroids play a key role in mood regulation by modulating the GABAergic system. The reduction in allopregnanolone is hypothesized to impair emotional stability, increasing the risk of depression in susceptible individuals [20]. A study by Welk et al. involving 93,197 men aged 66 years and older found a 94% increased risk of depression during the first 18 months of finasteride use (HR, 1.94; 95% CI, 1.73–2.16). Notably, while the risk declined over time, it remained slightly elevated throughout the follow-up period . Similarly, the same study reported a significant increase in anxiety-related diagnoses among users of finasteride. A smaller study involving 1,204 men taking finasteride for androgenetic alopecia found that 2.4% of participants developed new-onset depression, with symptoms resolving in most cases within six months after discontinuation. This highlights the potential reversibility of finasteride-induced mood changes [21]. A pharmacovigilance study analyzing the World Health Organization’s global database found a significant signal for depression in finasteride users under the age of 45 (ROR, 4.33; 95% CI, 4.17–4.49). This finding emphasizes the potential vulnerability of younger men to the drug’s psychiatric effects [4].

**Dementia and Alzheimer’s Disease**

Some studies have indicated a potential association between finasteride use and an elevated risk of dementia, including Alzheimer’s disease. Hypothetically, this would be due to the reduction in neurosteroids such as allopregnanolone, which may play a key role in protecting against neurodegeneration by promoting synaptic plasticity, reducing neuroinflammation, and preventing the accumulation of beta-amyloid plaques. A decrease in these neurosteroids could lead to cognitive impairments and an increased risk of neurodegenerative diseases [22].

In one Swedish cohort study involving 2,236,876 men aged 50–90 years, the results indicated a 61% increased risk of developing dementia among finasteride users, with a hazard ratio of 1.61 for all-cause dementia. Noteworthy, the study also reported an even higher risk for Alzheimer's disease, with a hazard ratio of 1.85, suggesting a stronger association between finasteride and this specific condition. Similarly, a study conducted on a Danish cohort of 1,983,785 individuals, followed for a median of 10 years, observed a potential link between the use of finasteride and the risk of Alzheimer's disease. In this cohort, an elevated risk of Alzheimer's disease was reported among finasteride users, although the overall association with all-cause dementia appeared less pronounced [23].

A large-scale Canadian study further investigated the risk of dementia associated with 5-alpha reductase inhibitors (5ARIs), including finasteride and dutasteride, in a matched cohort of 81,162 men aged 66 years and older. During the first year of 5ARI use, the hazard ratio for developing dementia was 2.18 (95% CI: 2.01–2.35), indicating a more than twofold increase compared to non-users. The risk decreased to 1.52 (95% CI: 1.39–1.67) during the second year of use and was not statistically significant in long-term users (HR: 1.06; 95% CI: 0.98–1.14). The study found no significant differences between finasteride and dutasteride in terms of dementia risk. These findings suggest that the observed early increase in dementia diagnoses may be attributable to lead-time bias, where men with pre-clinical dementia are more likely to seek treatment for urinary symptoms, leading to earlier detection of cognitive impairment [24].

**Suicidal ideation**

In the scientific literature, concerns regarding suicidal ideation (SI) linked to finasteride use first emerged prominently in the early 2010s. These concerns were based on increasing case reports and small studies suggesting an association between finasteride, a 5-alpha reductase inhibitor (5-ARI), and psychological adverse events, including suicidal thoughts and behaviors

In 2020, a pharmacovigilance study using VigiBase, the World Health Organization's global database of adverse drug reactions, analyzed 356 reports of suicidality and 2,926 psychological adverse events related to finasteride use. The study found a significant disproportionality signal for suicidality (reporting odds ratio [ROR]: 1.63; 95% CI: 1.47–1.81), driven primarily by reports of suicidal ideation (ROR: 4.39; 95% CI: 3.90–4.95). Notably, this association was more pronounced in younger patients aged under 45 years and those using finasteride for androgenetic alopecia. In contrast, no increased risk of suicidality was observed in older patients with benign prostatic hyperplasia [4].

Similarly, a 2015 retrospective study using the U.S. Food and Drug Administration's Adverse Event Reporting System (FAERS) evaluated 39 cases of suicidal ideation among men aged 18–45 years treated with low-dose finasteride for androgenic alopecia. In this study, 87% of individuals reporting suicidal ideation also experienced persistent sexual dysfunction (SD), suggesting a potential interplay between these adverse outcomes. Although suicidal ideation did not meet the formal safety signal threshold in this analysis (empirical Bayes geometric mean [EBGM]: 1.72; 95% CI: 1.31–2.23), the findings underscored the need for further investigation [5].

**Prostate Cancer**

In the scientific literature, as early as the 2000s, studies emerged highlighting the dual effects of 5-alpha reductase inhibitors (5-ARIs), such as finasteride, on prostate cancer risk. Finasteride was designed to treat benign prostatic hyperplasia (BPH), was also hypothesized to prevent prostate cancer due to their ability to inhibit the conversion of testosterone to dihydrotestosterone (DHT), a key androgen in prostate tissue growth. However, subsequent evidence raised concerns about the potential for 5-ARIs to increase the risk of high-grade prostate cancer while reducing the incidence of low-grade tumors [25].

The Prostate Cancer Prevention Trial (PCPT), a large randomized study involving 18,882 men aged 55 and older, reported that finasteride reduced the overall incidence of prostate cancer by 25%. However, the trial also observed a 27% relative increase in the risk of high-grade prostate cancers (Gleason score ≥7) among finasteride users compared to placebo. This finding raised concerns about the safety of finasteride as a chemopreventive agent. Subsequent analyses suggested that the increased detection of high-grade tumors may be partly due to the prostate volume reduction caused by finasteride, which enhances biopsy sensitivity and facilitates the identification of smaller, aggressive tumors .[2]

Mechanistic insights suggest that finasteride effectively suppresses low-grade prostate cancer cells reliant on DHT while potentially creating an environment that allows high-grade cancer cells, which may be less

dependent on androgens, to proliferate. This hypothesis aligns with findings from multiple studies indicating an increased risk of high-grade tumors in finasteride users [16].

An analysis published in the New England Journal of Medicine noted that while finasteride reduced the relative risk of Gleason 6 or lower prostate cancer, it was associated with an absolute increase of 0.7% in the incidence of tumors with Gleason scores of 8–10. This translates to one additional high-grade cancer diagnosis for every 150 to 200 men treated with finasteride. The study emphasized the complexity of balancing the reduced risk of low-grade cancers against the increased likelihood of high-grade cancers [26]

**Male Breast Cancer**

Male breast cancer is an exceptionally rare disease, with an incidence rate of approximately 1 case per 100,000 person-years. Despite the rarity of this condition, initial case reports raised questions about a possible association, prompting several investigations.

A 2009 review by the UK Medicines and Healthcare Products Regulatory Agency (MHRA) summarized 50 worldwide cases of male breast cancer in patients treated with 5 mg finasteride for benign prostatic hyperplasia (BPH). The median time to cancer onset was reported as approximately 36 months after initiating finasteride therapy. However, the lack of robust epidemiological studies and potential underreporting made it difficult to establish causality. Notably, three additional cases of male breast cancer were associated with the 1 mg dose of finasteride used for androgenic alopecia, but the short duration of exposure in these cases was deemed insufficient to support a causal relationship [27].

A more recent Nordic population-based case-control study analyzed data from Denmark, Finland, and Sweden. This study included 1,005 male breast cancer cases and 43,058 controls. Adjusting for confounders, the odds ratio (OR) for finasteride use and male breast cancer was 1.09 (95% CI: 0.77–1.54), suggesting no significant association. Sensitivity analyses examining cumulative finasteride exposure also found no evidence of a dose-response relationship, and subgroup analyses yielded similar results [28].

Similarly, a large U.S.-based case-control study with 339 cases of male breast cancer and 6,780 matched controls reported no statistically significant associations between finasteride exposure and male breast cancer. Even with extended cumulative exposure, the relative risk (RR) estimates remained close to null (RR for ≥3 years of exposure: 0.75; 95% CI: 0.27–2.10). The proposed mechanism for a potential link involves changes in the estrogen-to-testosterone ratio caused by finasteride. By inhibiting dihydrotestosterone (DHT), finasteride could lead to a relative increase in estrogen activity, which may promote the development of hormone-sensitive malignancies like breast cancer. However, evidence from both preclinical and epidemiological studies has failed to substantiate this mechanism, and the observed cases may reflect surveillance bias or random variation rather than a true pharmacological effect [29].

**Gynecomastia**

Gynecomastia, characterized by benign enlargement of glandular breast tissue in males, is a rare but documented adverse effect associated with finasteride use. It has been reported in patients taking both the 5 mg dose for benign prostatic hyperplasia (BPH) and the 1 mg dose for androgenetic alopecia. The condition often manifests as unilateral or bilateral breast enlargement, which can be accompanied by tenderness or discomfort. The mechanism behind the development of gynecomastia involves the inhibition of the enzyme 5α-reductase type II by finasteride, leading to decreased conversion of testosterone to DHT. This results in an increase in serum testosterone levels, some of which are aromatized to estrogen, thereby increasing estrogen levels relative to androgens. Elevated estrogen levels promote the proliferation of glandular breast tissue, contributing to the development of gynecomastia in susceptible individuals . [30]

In a study analyzing adverse event reports in the FDA's FAERS database from 2000 to 2019, Harrell et al. identified gynecomastia as one of the reported adverse effects associated with finasteride use. Among cases of monotherapy with finasteride at doses of 1 mg and 5 mg, gynecomastia was a frequent concern, although the incidence varied depending on the dosage and patient population [31].

In a cohort study by Rossi et al., conducted over a 10-year period, gynecomastia was reported in approximately 6% of men treated with finasteride at 1 mg/day for androgenetic alopecia. Despite this, some patients continued

therapy due to the drug's benefits in hair growth. The risk appeared to be dose-dependent, with lower doses associated with a reduced incidence compared to the 5 mg dose used for benign prostatic hyperplasia [32].

A cohort study using the UK Clinical Practice Research Datalink evaluated the risk of gynecomastia in men with benign prostatic hyperplasia (BPH) treated with finasteride. The study reported that the incidence rate of gynecomastia was significantly higher in finasteride users compared to unexposed men. Specifically, the incidence rate ratio (IRR) for gynecomastia in finasteride users was 3.55 (95% CI: 3.05–4.14). This increased risk remained consistent across various treatment durations and was not significantly affected by other medications associated with gynecomastia [33].

**Conclusion**

Finasteride remains a cornerstone treatment for benign prostatic hyperplasia (BPH) and androgenetic alopecia (AGA), with a well-documented efficacy in reducing prostate volume and promoting hair regrowth. However, its use is not without risks. Adverse effects such as sexual dysfunction, gynecomastia, and neuropsychiatric risks, including depression and suicidal ideation, underscore the importance of patient education and monitoring during therapy. The evidence highlights that most adverse effects are mild and reversible upon discontinuation, though a subset of patients may experience persistent side effects, particularly sexual dysfunction. Finasteride also offers additional benefits, including a reduction in the risk of low-grade prostate cancer in older men. However, the increased detection of high-grade tumors raises questions about its safety profile in this context. Its impact on spermatogenesis and fertility is rare and transient but warrants caution in men with reproductive concerns. In conclusion, finasteride provides significant therapeutic value when prescribed judiciously, with careful attention to patient selection and ongoing monitoring. Future studies should aim to further elucidate its long-term safety, particularly in younger populations, and to better stratify patients based on their risk of adverse effects. This approach will help optimize the balance between finasteride's benefits and risks, ensuring its continued role in clinical practice.

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Author's contribution:

Conceptualization and Methodology: AK, AD, AS; Software: Not applicable; Check: WW, MK, MMJ; Formal analysis: AK, AS, BK, AKG; Investigation: AD, MM, MK; Resources: Not applicable; Writing - rough preparation: AK, AD, MMJ, BK, WW, NR; Writing - review and editing: MMJ, MM, NR; Visualization: BK, AD, MMJ, WW; Supervision: AK, AD, AKG; Project administration: AK.

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