
**International Journal of Innovative Technologies in Social Science**

e-ISSN: 2544-9435

**Operating Publisher**
**SciFormat Publishing Inc.**
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

| | |
|---|---|
| **ARTICLE TITLE** | FINASTERIDE IN MODERN MEDICINE: THERAPEUTIC APPLICATIONS AND SYSTEMIC HEALTH IMPLICATIONS |
| **DOI** | https://doi.org/10.31435/ijitss.1(49).2026.4993 |
| **RECEIVED** | 19 January 2026 |
| **ACCEPTED** | 26 March 2026 |
| **PUBLISHED** | 30 March 2026 |
| **LICENSE** | The article is licensed under a **Creative Commons Attribution 4.0 International License.** |

# FINASTERIDE IN MODERN MEDICINE: THERAPEUTIC APPLICATIONS AND SYSTEMIC HEALTH IMPLICATIONS

***Aleksandra Kutaj*** *(Corresponding Author, Email: Ola.kutaj1@gmail.com)*
*Poznan University of Medical Sciences, Poznań, Poland*
*ORCID ID: 0009-0001-5145-0752*

***Wiktor Łapa***
*Poznan University of Medical Sciences, Poznań, Poland*
*ORCID ID: 0009-0000-8806-2713*

***Tatyana Savitskaya***
*Grodno State Medical University, Hrodna, Belarus*
*ORCID ID: 0009-0008-8728-7329*

***Maria Sobolewska***
*Jagiellonian University Medical College, Kraków, Poland*
*ORCID ID: 0009-0000-0036-7070*

***Olga Magdalena Tuleja***
*Jagiellonian University Medical College, Kraków, Poland*
*ORCID ID: 0009-0005-7904-7323*

***Dawid Wiczkowski***
*Independent Public Specialist Western Hospital of John Paul II in Grodzisk Mazowiecki, Grodzisk Mazowiecki, Poland*
*ORCID ID: 0009-0004-7050-9598*

**ABSTRACT**

Finasteride, a 5α-reductase inhibitor, is widely used for the treatment of androgenetic alopecia and benign prostatic hyperplasia. Its ability to reduce dihydrotestosterone (DHT) levels and promote hair growth has been well documented, making it a common therapeutic option among men. However, growing attention has been given to reports of persistent adverse effects affecting sexual, psychological, and metabolic health, which require further investigation. This review summarizes current findings on the dermatological and systemic effects of finasteride, emphasizing the need for comprehensive risk assessment and continued research. The literature review was conducted using the PubMed database, analyzing English-language studies published mainly between 2023 and 2025, identified with the keywords „finasteride", „androgenetic alopecia", „post-finasteride syndrome" and „adverse effects". Despite these concerns, finasteride remains a clinically valuable and frequently prescribed medication for both androgenetic alopecia and benign prostatic hyperplasia. Reported persistent symptoms highlight the importance of proper patient education, informed consent, and individualized therapy. Further studies are required to clarify the mechanisms, prevalence, and management of these effects, ensuring safer and more informed clinical use of finasteride in both therapeutic and aesthetic contexts.

**KEYWORDS**

Finasteride, Androgenic Alopecia, Drug-Related Side Effects and Adverse Reactions, Health Behavior, Modern Civilization

**CITATION**

Aleksandra Kutaj, Wiktor Łapa, Tatyana Savitskaya, Maria Sobolewska, Olga Magdalena Tuleja, Dawid Wiczkowski. (2026) Finasteride in Modern Medicine: Therapeutic Applications and Systemic Health Implications. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.4993

## Introduction

Finasteride is a 5α-reductase inhibitor with a greater affinity for the type 2 isoenzyme while also inhibiting type 1. These enzymes mediate the conversion of testosterone into DHT, a key factor in the development of androgenetic alopecia (AGA) in genetically predisposed individuals and benign prostatic hyperplasia (BPH) [1–4].

Due to its ability to reduce DHT levels, finasteride has become one of the most commonly prescribed pharmacological treatments for this condition. Initially approved by the U.S. Food and Drug Administration in 1992 for the treatment of BPH, it was introduced in 1997 for AGA male pattern at a daily dose of 1 mg. Clinical studies have demonstrated its effectiveness in slowing hair loss progression and even promoting hair regrowth [5–7].

Although 5α-reductase inhibitors such as finasteride are generally considered well-tolerated and relatively safe, emerging research has explored aspects of their long-term safety profile [8]. While finasteride remains a cornerstone treatment for both BPH and AGA, its use has been associated with a range of adverse effects, some of which have been reported by patients even after discontinuation of the drug. Reported persistent adverse effects include sexual dysfunction, mood disturbances, cognitive impairments, fatique and dermatological manifestations [1].

Some researchers emphasize that the concept of the interpretation of persistent symptoms reported after finasterid remains a matter of debate [9]. The literature also points to the possible contribution of the nocebo effect, in which symptoms may arise from patients' negative expectations rather than from the direct pharmacological action of the drug. Therefore, in the assessment of symptoms attributed to finasteride, it is important to consider not only biological mechanisms but also the influence of patients' expectations [8].

Owing to these concerns, regulatory agencies in multiple countries have issued communications regarding the potential long-term risks associated with finasteride and other 5α-reductase inhibitors. As finasteride remains a prevalent treatment for androgenetic alopecia, ongoing research is necessary to reassess the balance between its benefits and potential risks. A deeper understanding of its long-term safety profile is crucial to optimizing patient care and minimizing adverse effects [9]. Given the growing popularity of finasteride for aesthetic purposes among young men, this issue reflects broader challenges of modern civilization, where lifestyle-related medical interventions increasingly influence overall health.

## Aim of the work

This review provides an overview of current evidence on the dermatological and systemic effects of finasteride, highlighting the importance of comprehensive risk assessment and ongoing research.

## Methods

The literature review was conducted using the PubMed database to identify recent studies related to finasteride and its systemic effects. The search included the keywords „finasteride", „5α-reductase inhibitor", „androgenetic alopecia", „post-finasteride syndrome", „adverse effects", combined with operators OR and AND. Publications mainly from 2023–2025, with reference to earlier relevant studies (2000–2022), were analyzed. The final analysis included 36 studies, which were used to summarize current evidence on the therapeutic role and safety profile of finasteride.

## Literature review results

### The impact of finasteride on skin and hair

*Reduction of DHT in scalp and serum*

Finasteride significantly reduces DHT concentrations in both scalp tissue and serum, which correlates with improved hair growth and stabilization of androgenetic alopecia. In a clinical study involving 1087 men, scalp DHT levels decreased by approximately 64% following finasteride treatment. Recent research also highlights the benefits of topical formulations, which maintain therapeutic efficacy while reducing systemic DHT suppression. In a Phase III randomized controlled trial, topical finasteride spray (50–200 μL/day) significantly increased hair density after 24 weeks compared with placebo, with adverse effects limited mainly to mild scalp irritation or pruritus [10].

These findings suggest that formulations or dosing strategies minimizing systemic DHT suppression may help maintain therapeutic efficacy while reducing the likelihood of treatment-related adverse events.

*Effects on Hair Structure and Growth*

Beyond biochemical changes, the clinical efficacy of finasteride is reflected in measurable improvements in hair density and hair growth dynamics. A daily dose of 1 mg finasteride significantly increases total hair count compared to placebo. Studies have reported an average increase of 12.4 hairs/cm² after 24 weeks and 16.4 hairs/cm² after 48 weeks of therapy [9]. Similar results have been observed with topical application of finasteride, where the increase in hair count in the target area after 24 weeks was significantly greater compared to placebo; however, inhibition of DHT activity with topical finasteride is less pronounced than with the oral formulation. Moreover, topical finasteride exhibited better systemic tolerance and had a lesser impact on serum DHT levels [11,12].

Beyond increasing total hair count, finasteride also significantly affects the hair growth cycle by increasing the number of hairs in the anagen phase and improving the anagen-to-telogen ratio. A randomized study involving 212 men aged 18–40 years with androgenetic alopecia showed that 1 mg of finasteride daily for 48 weeks led to a significant increase in both total hair count and anagen-phase hair count. At the start of the study, the mean total hair count and anagen-phase hair count in the finasteride group were 200 and 124 hairs, respectively (% anagen = 62%), with an anagen-to-telogen ratio of 1.74. After 48 weeks, finasteride treatment resulted in an improvement of total hair count by 17.3 ± 2.5 hairs (8.3% ± 1.4%) and anagen-phase hair count by 27.0 ± 2.9 hairs (26% ± 3.1%). Additionally, finasteride treatment improved the anagen-to-telogen ratio by 47% [13].

These findings provide direct evidence that finasteride promotes the transition of hairs into the anagen phase, contributing to visible hair growth improvement in treated patients.

### Persistent adverse effects potentially associated with finasteride

A growing number of studies have described a set of persistent symptoms affecting multiple physiological and psychological functions. These symptoms may persist even after discontinuation of treatment and include a wide range of ailments, such as sexual dysfunction, mood disorders, metabolic disturbances, dermatological issues, and cardiovascular complications [9,14–17].

*Pathophysiology*

Persistent symptoms reported after finasteride use have been hypothesized to be associated with disturbances in the metabolism of neuroactive steroids and dysfunction of hormonal axes. Finasteride, as a 5α-reductase inhibitor, blocks the conversion of testosterone to DHT and progesterone to allopregnanolone (THP) [18–20].

DHT and THP, classified as neuroactive steroids, are essential not only for the normal functioning of androgen target organs such as the prostate gland and the skin but also for maintaining neurochemical homeostasis within the central nervous system. These steroids act on both classical steroid receptors (androgen and progesterone receptors) and non-classical receptors, including GABA-A receptors. Given their role in the modulation of neurotransmission, a hypothesis has been proposed that a reduction in neuroactive steroid levels may contribute to the development of depressive symptoms observed following finasteride treatment [21].

Some studies suggest that, finasteride may influence gene expression, leading to androgen receptor (AR) upregulation, increased histone acetylation, and alterations in the methylation status of multiple receptors and enzymes. Such epigenetic modifications may disrupt neurotransmitter systems, including the dopaminergic system, potentially contributing to the persistence of symptoms such as anxiety, depression, and suicidal ideation [22].

Therefore, disturbances in neuroactive steroid metabolism and dysfunction of neurotransmitter systems have been proposed as potential mechanisms underlying the persistent symptoms reported after finasteride use. While these hypotheses offer valuable insight, the biological basis of persistent symptoms following finasteride use remains to be confirmed through large-scale, controlled studies.

**Clinical manifestations**

*Sexual dysfunction*

The range of adverse effects, particularly in the context of sexual dysfunction, was described in a study by Harrell et al., which compared a group of 3,266 patients taking finasteride 1 mg with 744 patients taking 5 mg. This study reported that such adverse effects were observed across different doses and are especially prevalent among younger patients. Patients taking 1 mg of finasteride most commonly reported adverse effects related to libido (40%), erectile function (51%), orgasm (5%), and ejaculation (24%). What is more, 13% of patients reported various penile-related complications, including a perceived reduction in penile size, structural abnormalities such as curvature or deformity, localized pain, diminished genital sensation [23].

Similar sexual disturbances are reported in the study by Chiriacò et al., where the ASEX questionnaire revealed that 40.5% of patients had difficulty maintaining erections, and 3.8% were unable to achieve one. Additionally, 16.5% reported trouble reaching orgasm, with 2.5% unable to do so. In a study-specific questionnaire, the most frequent symptoms were reduced penile sensitivity (87.3%), decreased ejaculatory force (82.3%), and lower penile temperature (78.5%) [24].

In 2022, a study by Li et al. was conducted, analyzing adverse effects, including sexual ones, in three male patients, aged 20 to 30 years, who had experienced PFS for a duration of 1 to 3 years. All patients experienced erectile dysfunction, and two of them reduction in genital size. What is more, one of them also reported testicular pain, low libido, and disturbances in orgasm and ejaculation [25].

It is worth noting that a study by Lauck et al. emphasizes that the risk of sexual dysfunction may be influenced by comorbidities such as hypertension, diabetes, obesity, depression and smoking. These findings may support a hypothesis regarding the persistence of sexual dysfunction after stopping 5-ARIs [26].

The severity of these symptoms can profoundly affect patients' quality of life, leading to significant emotional and psychological burdens. Given their persistent nature and the limited treatment options available, persistent sexual dysfunction reported after finasteride use remains a major clinical challenge requiring further research and therapeutic strategies.

*Mood Disorders*

Mood disorders have been frequently reported by patients describing persistent symptoms after finasteride use including depression, anxiety, and suicidal thoughts [9,18]. A growing body of evidence suggests an association between finasteride use and an increased risk of mood disturbances**.** In a large cohort of 23,227 men treated for androgenetic alopecia (mean age 31.4 ± 10.3 years) compared to 39,444 controls, psychiatric disorders occurred in 1% of finasteride users. Rates of anxiety (0.6% vs. 0.4%, $p = 0.04$) and depression (0.5% vs. 0.4%, $p = 0.007$) were significantly higher among patients receiving finasteride. Multivariate regression confirmed finasteride as an independent risk factor for anxiety (OR 1.449, $p = 0.002$) and depression (OR 1.439, $p = 0.003$) [27].

Recent data from France indicate that almost 40% of men treated with finasteride for androgenetic alopecia reported suicidal thoughts or attempts, while over 60% of psychiatric adverse reactions were classified as severe. These depressive symptoms and suicidality often appeared in individuals without previous psychiatric disorders or sexual dysfunction, and in many cases persisted for more than 20 months after discontinuing the treatment [28].

In addition persistent symptoms reported after finasteride use have included cognitive impairments and emotional dysregulation. A retrospective analysis of medical data from 2016, involving 79 men aged 18–50 years, identified anhedonia- the inability to experience pleasure-as the most common non-sexual symptom, affecting 75.9% of participants. Furthermore, 72.2% of individuals reported concentration difficulties, which may contribute to diminished quality of life, occupational performance, and social functioning [25].

The psychiatric and cognitive symptoms reported in some patients after finasteride use highlight the need for further research into the underlying neurobiological mechanisms. Studies by Traish (2020) suggest that alterations in neuroactive steroid levels may contribute to these adverse effects, warranting further investigation into potential therapeutic interventions [9].

These findings underscore the significant psychological impact of finasteride use, reinforcing the necessity for continued research and the development of effective support strategies for affected individuals.

*Metabolic and Cardiovascular Events*

The possible association between finasteride use and alterations in glucose metabolism, including type 2 diabetes, has been explored in several studies. Wei et al. analyzed a cohort of 5890 individuals taking finasteride, with data sourced from both the CPRD (3445 individuals) and NHIRD (2445 individuals) databases. Their study reported a moderate but statistically significant association between finasteride use and type 2 diabetes risk [29].

Further evidence supporting the potential impact of finasteride on glucose metabolism is provided by a case report by Bose et al. They described a 69-year-old male patient who experienced significant deterioration in glycemic control following the initiation of finasteride therapy. Despite treatment with a GLP-1 receptor agonist, SGLT2 inhibitors, and NPH insulin, the patient's blood glucose levels markedly increased, with hemoglobin A1c rising from 7% to 8.2%. Additionally, weight gain was observed during finasteride use. This case report suggest a possible temporal relationship between finasteride use and changes in glicemic control, supporting the need for further research into potential metabolic effects [30].

In addition to disturbances in glucose metabolism, effects on lipid profiles have been reported. McQueen et al. analyzed retrospective data from the NHANES database and found that individuals using finasteride had lower levels of total cholesterol and LDL-C compared to non-users, with no significant differences in HDL-C levels. These findings were limited to individuals previously diagnosed with hypercholesterolemia or receiving statin therapy [31].

Beyond metabolic effects, some authors have suggested that finasteride, through inhibition of 5α-reductase and subsequent alterations in steroid metabolism, may effect cardiovascular regulation. By affecting glucocorticoid and mineralocorticoid clearance, finasteride could promote vascular dysfunction, endothelial impairment, and alterations in blood pressure regulation. Although current clinical data remain limited, these potential cardiovascular consequences highlight the need for long-term studies assessing the safety profile of finasteride, not only in the context of metabolic health but also broader cardiovascular outcomes [1].

**Treatment Strategies for Finasteride-Related Adverse Effects**

Management of persistent symptoms reported after finasteride use remains a considerable clinical challenge, as no therapies with proven efficacy have been established to date. Current management strategies primarily focus on symptomatic relief, addressing manifestations such as sexual dysfunction, mood disturbances, and anxiety. Therapeutic approaches should be individualized and carefully tailored to the patient's specific clinical presentation [10]. In cases where neuropsychiatric symptoms predominate, standard treatments for mood disorders, including pharmacotherapy and psychotherapy, are recommended [9].

Experimental therapeutic options have also been proposed. Case reports in the literature describe the use of testosterone replacement therapy, human chorionic gonadotropin (hCG), and hydrocortisone; however, due to the lack of controlled studies confirming their efficacy, these interventions remain theoretical and warrant further research [32].

Additionally, some studies have suggested that patients reporting persistent symptoms after finasteride use may exhibit alterations in gut microbiota composition, including reduced bacterial diversity and decreased abundance of beneficial strains such as *Faecalibacterium*. Modulation of the gut microbiome, for instance through probiotic therapy, is being explored as a potential future therapeutic avenue [33–36].

Finally, genetic studies have reported variations in genes such as *HLA-B*, *CA8*, and *VSIG10L2* among patients describing persistent symptoms after finasteride use. These findings raise the possibility of implementing prophylactic genetic screening prior to the initiation of finasteride therapy, although further validation in larger cohorts is necessary [26].

**Conclusions**

Finasteride, a 5α-reductase inhibitor, is widely employed in the treatment of androgenetic alopecia and benign prostatic hyperplasia by lowering dihydrotestosterone (DHT) levels. By inhibiting DHT production, it effectively slows the progression of hair loss and promotes regrowth by prolonging the anagen phase of the hair cycle. Although considered clinically effective, *reports of persistent adverse effects following finasteride use have prompted increasing research interest into its long-term safety.* A subset of patients has reported persistent sexual, cognitive, metabolic, and dermatological symptoms following finasteride discontinuation, though the pathophysiological basis of these effects remains unclear. Current treatment strategies are largely limited to symptomatic management, with pharmacological and psychological interventions addressing the most debilitating manifestations. Experimental therapies, including hormone modulation, gut microbiota

targeting, and agents reversing epigenetic changes, are being explored but require validation through controlled clinical trials. As finasteride remains a widely prescribed therapeutic option, ongoing research is crucial to better understand its long-term risks and to establish effective, evidence-based strategies for the prevention and management for the prevention and management of finasteride-associated adverse effects. The clinical relevance and biological mechanisms underlying these reported symptoms remain debated, highlighting the need for further well-designed studies.

**Disclosures and acknowledgements**

The authors declare no conflicts of interest with respect to the research, authorship, and/or publication of this article. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Artificial intelligence (AI) was not used in the creation of the manuscript.

## REFERENCES

1. Cilio, S., Tsampoukas, G., Morgado, A., et al. (2025). Post-finasteride syndrome—A true clinical entity? *International Journal of Impotence Research, 37*, 426–435. https://doi.org/10.1038/s41443-025-01025-6
2. Meenrajan, S. R. (2024). Using 5 alpha reductase inhibitors safely: What primary care physicians need to know. *Journal of Family Medicine and Primary Care, 13*, 4797–4799. https://doi.org/10.4103/jfmpc.jfmpc_575_24
3. Ntshingila, S., Oputu, O., Arowolo, A. T., et al. (2023). Androgenetic alopecia: An update. *JAAD International, 13*, 150–158. https://doi.org/10.1016/j.jdin.2023.07.005
4. Panuganti, V. K., Madala, P. K., Grandhi, V. R., et al. (2025). A randomized, double-blind, placebo and active controlled phase II study to evaluate the safety and efficacy of novel dutasteride topical solution (0.01%, 0.02%, and 0.05% w/v) in male subjects with androgenetic alopecia. *Cureus*. https://doi.org/10.7759/cureus.89309
5. Kaiser, M., Abdin, R., Gaumond, S. I., et al. (2023). Treatment of androgenetic alopecia: Current guidance and unmet needs. *Clinical, Cosmetic and Investigational Dermatology, 16*, 1387–1406. https://doi.org/10.2147/CCID.S385861
6. Kim, J., Song, S.-Y., & Sung, J.-H. (2025). Recent advances in drug development for hair loss. *International Journal of Molecular Sciences, 26*, 3461. https://doi.org/10.3390/ijms26083461
7. Santana, F. D. F. V., Lozi, A. A., Gonçalves, R. V., et al. (2023). Comparative effects of finasteride and minoxidil on the male reproductive organs: A systematic review of in vitro and in vivo evidence. *Toxicology and Applied Pharmacology, 478*, 116710. https://doi.org/10.1016/j.taap.2023.116710
8. Brezis, M. (2025). Failing public health again? Analytical review of depression and suicidality from finasteride. *Journal of Clinical Psychiatry, 86*. https://doi.org/10.4088/JCP.25nr15862
9. Traish, A. M. (2020). Post-finasteride syndrome: A surmountable challenge for clinicians. *Fertility and Sterility, 113*, 21–50. https://doi.org/10.1016/j.fertnstert.2019.11.030
10. Chen, S., Li, L., Ding, W., et al. (2025). Androgenetic alopecia: An update on pathogenesis and pharmacological treatment. *Drug Design, Development and Therapy, 19*, 7349–7363. https://doi.org/10.2147/DDDT.S542000
11. Keerti, A., Madke, B., Keerti, A., et al. (2023). Topical finasteride: A comprehensive review of androgenetic alopecia management for men and women. *Cureus*. https://doi.org/10.7759/cureus.44949
12. Piraccini, B. M., Blume-Peytavi, U., Scarci, F., et al. (2022). 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, 36*, 286–294. https://doi.org/10.1111/jdv.17738
13. Van Neste, D., Fuh, V., Sanchez-Pedreno, P., et al. (2000). Finasteride increases anagen hair in men with androgenetic alopecia. *British Journal of Dermatology, 143*, 804–810. https://doi.org/10.1046/j.1365-2133.2000.03780.x
14. Low, P., Li, K. D., Hakam, N., et al. (2022). 5-alpha reductase inhibitor related litigation: A legal database review. *Andrology, 10*, 470–476. https://doi.org/10.1111/andr.13145
15. Zhong, X., Yang, Y., Wei, S., et al. (2025). Multidimensional assessment of adverse events of finasteride: A real-world pharmacovigilance analysis based on FDA Adverse Event Reporting System (FAERS) from 2004 to April 2024. *PLOS ONE, 20*, e0309849. https://doi.org/10.1371/journal.pone.0309849
16. Zhang, H., Ou, H., Zhao, P., et al. (2025). Association between finasteride with subjective memory deficits: A study from the NHANES and FAERS databases. *Frontiers in Neurology, 16*, 1616851. https://doi.org/10.3389/fneur.2025.1616851
17. Leliefeld, H. H. J., Debruyne, F. M. J., & Reisman, Y. (2025). The post-finasteride syndrome: Possible etiological mechanisms and symptoms. *International Journal of Impotence Research, 37*, 414–421. https://doi.org/10.1038/s41443-023-00759-5

18. Xia, Y., Chen, H., Chen, Y., et al. (2025). Relative efficacy of minoxidil in combination with other treatments for androgenic alopecia: A network meta-analysis based on randomized controlled trials. *Frontiers in Medicine, 12*, 1638496. https://doi.org/10.3389/fmed.2025.1638496
19. Johnson, H., Huang, D., Clift, A. K., et al. (2025). Effectiveness of combined oral minoxidil and finasteride in male androgenetic alopecia: A retrospective service evaluation. *Cureus*. https://doi.org/10.7759/cureus.77549
20. Diviccaro, S., Oleari, R., Amoruso, F., et al. (2025). Exploration of the possible relationships between gut and hypothalamic inflammation and allopregnanolone: Preclinical findings in a post-finasteride rat model. *Biomolecules, 15*, 1044. https://doi.org/10.3390/biom15071044
21. Maguire, J. L., & Mennerick, S. (2024). Neurosteroids: Mechanistic considerations and clinical prospects. *Neuropsychopharmacology, 49*, 73–82. https://doi.org/10.1038/s41386-023-01626-z
22. Melcangi, R. C., Santi, D., Spezzano, R., et al. (2017). Neuroactive steroid levels and psychiatric and andrological features in post-finasteride patients. *Journal of Steroid Biochemistry and Molecular Biology, 171*, 229–235. https://doi.org/10.1016/j.jsbmb.2017.04.003
23. Harrell, M. B., Ho, K., Te, A. E., et al. (2021). An evaluation of the federal adverse events reporting system data on adverse effects of 5-alpha reductase inhibitors. *World Journal of Urology, 39*, 1233–1239. https://doi.org/10.1007/s00345-020-03314-9
24. Chiriacò, G., Cauci, S., Mazzon, G., et al. (2016). An observational retrospective evaluation of 79 young men with long-term adverse effects after use of finasteride against androgenetic alopecia. *Andrology, 4*, 245–250. https://doi.org/10.1111/andr.12147
25. Li, X., Guo, Y., Lu, Y., et al. (2022). Case report: A study of the clinical characteristics and genetic variants of post-finasteride syndrome patients. *Translational Andrology and Urology, 11*, 1452–1457. https://doi.org/10.21037/tau-22-92
26. Lauck, K. C., Limmer, A., Harris, P., et al. (2024). Sexual dysfunction with 5-alpha-reductase inhibitor therapy for androgenetic alopecia: A global propensity score matched retrospective cohort study. *Journal of the American Academy of Dermatology, 91*, 163–166. https://doi.org/10.1016/j.jaad.2024.03.019
27. Lyakhovitsky, A., Amichai, B., Galili, E., et al. (2024). The risk of psychiatric disorders in finasteride users with benign prostatic hyperplasia and androgenetic alopecia: A population-based case-control study. *Australasian Journal of Dermatology, 65*, 621–629. https://doi.org/10.1111/ajd.14359
28. Géniaux, H., & Laroche, M.-L. (2025). Isolated depressive disorders and suicidality with finasteride use for androgenetic alopecia: A call for enhanced vigilance. *Therapies*. https://doi.org/10.1016/j.therap.2025.09.004
29. Wei, L., Lai, E. C.-C., Kao-Yang, Y.-H., et al. (2019). Incidence of type 2 diabetes mellitus in men receiving steroid 5α-reductase inhibitors: Population based cohort study. *BMJ*, l1204. https://doi.org/10.1136/bmj.l1204
30. Bose, V., & Santos Martinez, D. (2023). THU372 Finasteride induced hyperglycemia. *Journal of the Endocrine Society, 7*, bvad114.805. https://doi.org/10.1210/jendso/bvad114.805
31. McQueen, P., Molina, D., Pinos, I., et al. (2024). Finasteride delays atherosclerosis progression in mice and is associated with a reduction in plasma cholesterol in men. *Journal of Lipid Research, 65*, 100507. https://doi.org/10.1016/j.jlr.2024.100507
32. Solanki, P., Eu, B., Smith, J., et al. (2023). Physical, psychological and biochemical recovery from anabolic steroid-induced hypogonadism: A scoping review. *Endocrine Connections, 12*, e230358. https://doi.org/10.1530/EC-23-0358
33. Carrington, A. E., Maloh, J., Nong, Y., et al. (2023). The gut and skin microbiome in alopecia: Associations and interventions. *Journal of Clinical and Aesthetic Dermatology, 16*, 59–64.
34. Diviccaro, S., Giatti, S., Cioffi, L., et al. (2022). Gut inflammation induced by finasteride withdrawal: Therapeutic effect of allopregnanolone in adult male rats. *Biomolecules, 12*, 1567. https://doi.org/10.3390/biom12111567
35. Giatti, S., Diviccaro, S., Cioffi, L., et al. (2024). Post-finasteride syndrome and post-SSRI sexual dysfunction: Two clinical conditions apparently distant, but very close. *Frontiers in Neuroendocrinology, 72*, 101114. https://doi.org/10.1016/j.yfrne.2023.101114
36. An, J., Song, Y., Kim, S., et al. (2023). Alteration of gut microbes in benign prostatic hyperplasia model and finasteride treatment model. *International Journal of Molecular Sciences, 24*, 5904. https://doi.org/10.3390/ijms24065904