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Impact of Finasteride on Serum PSA Levels in Men With BPH: A Systematic Review

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ABSTRACT
Finasteride is the main therapy for benign prostatic hyperplasia (BPH) because it blocks 5-α reductase. It prevents testosterone from being converted into dihydrotestosterone. This mechanism prevents prostate enlargement and affects the regulation of prostate-specific antigen (PSA) levels. These pharmacological activities confer therapeutic advantages; nonetheless, they may influence the interpretation of PSA results in prostate cancer screening and the associated clinical decisions. The goal of this systematic review was to synthesize the available evidence regarding how finasteride affects total PSA, free PSA, prostate volume, and PSA density (PSAD), to provide a comprehensive understanding of its therapeutic effects. An exhaustive search of PubMed and ScienceDirect was performed for studies examining the relationship between finasteride use and serum PSA levels (August 2025). The review followed the PRISMA guidelines for systematic reviews and meta-analyses. Two reviewers independently applied strict eligibility criteria for participant inclusion and data extraction. Only nine of the initial 2,473 studies met all inclusion criteria. The findings demonstrated that finasteride consistently reduced total PSA levels by 30% to 50% and free PSA levels by up to 45%. Prostate volume and PSAD both decreased by approximately 5% to 20%. These consistent trends emphasize the importance of applying appropriate adjustment factors when interpreting PSA levels in men receiving finasteride therapy for BPH.

INTRODUCTION

Benign prostatic hyperplasia (*BPH*) is the most prevalent non-malignant condition affecting older males. This condition is known in the medical field as bladder outlet obstruction, lower urinary tract symptoms (*LUTS*), and *benign prostatic hyperplasia* (*BPH*) . Histologically, *BPH* refers to cellular alterations within the prostate, whereas *BPE* denotes glandular hypertrophy—often developing from histological *BPH*—and bladder outlet obstruction refers to the impediment of urine flow (Benign Prostatic Hyperplasia (BPH): Guideline Summary, 2023).

The clinical progression of *BPH* is characterized by the abnormal proliferation of stromal and epithelial cells within the prostatic transition zone, the anatomical region surrounding the urethra (Figg, 2015). This hyperplasia narrows the urethral lumen, making bladder emptying more difficult. Such obstruction can lead to *LUTS*, urinary retention, or infection due to residual urine in the bladder. If left untreated, the condition may result in long-term high-pressure urinary retention, which can be harmful and cause permanent structural and functional damage to the bladder's detrusor muscle (Patel & Parsons, 2014).

There are three approaches available for the management of *BPH*: observation, medication, and surgery (Arnold, 2023; Cumberbatch, 2025; Halawani, 2024a; Tacklind, 2010). Two categories of contributing factors exist—immutable factors such as age, family

history, and geographic location, and modifiable factors such as obesity. Research indicates that these variables significantly influence the onset and progression of the disease (Chughtai et al., 2016). Individuals with *BPH* may experience *LUTS* and bladder outlet obstruction due to both static and dynamic mechanisms (Halawani, 2024b; Sandhu, 2024; Schally, 2025). The enlarged prostate exerts pressure on the urethra and alters the contour of the bladder neck, thereby impeding urination (Foo, 2017). The dynamic mechanism involves increased smooth muscle tone in the prostate, accompanied by reduced elasticity and collagen content in the prostatic urethra, which exacerbates compliance issues and raises the risk of obstruction. This demonstrates that prostatic volume alone is an insufficient indicator of disease severity (Foo, 2017; Li et al., 2022; Page et al., 2021; Patel & Parsons, 2014; Salisbury et al., 2024).

The FDA has approved dutasteride and finasteride as *5α-reductase inhibitors* (*5-ARIs*) for the treatment of *BPH*. These medications inhibit the conversion of testosterone to dihydrotestosterone (*DHT*), a hormone critical to both prostate development and male-pattern hair loss. The clinical use of *5-ARIs* requires adherence to specific guidelines, including precise indications and monitoring for potential drug interactions (Chiu et al., 2023; Chughtai et al., 2016). Studies have shown that finasteride reduces *DHT* levels in the blood by up to 70% and in the prostate by more than 90%, regardless of dosage. Over time, such suppression decreases serum *PSA* levels by approximately 50% and prostate volume by about 25% (Salisbury, Leslie, & Tadi, 2024).

In this *systematic review*, a critical analysis is conducted of two previous studies on *BPH* and its management. The first study by Yu et al. (2020) evaluated the efficacy and side effects of commonly prescribed *BPH* medications, including finasteride and tamsulosin. It found that although these drugs effectively reduced symptoms, they were associated with side effects such as sexual dysfunction and ejaculatory disorders. The second study by Chiu et al. (2023) provided an updated overview of *BPH* management, including pharmacological therapies and minimally invasive surgical techniques such as *Aquablation* and *Urolift* therapy. This research emphasized the importance of an individualized approach in determining the optimal therapy for patients with *BPH*.

METHOD

This systematic review adhered to the guidelines established by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (Page et al., 2021). We utilized a systematic search approach based on a study topic developed using the PICO framework to explore the PubMed and ScienceDirect databases. The focus group was made up of men who had been diagnosed with benign prostatic hyperplasia (BPH) and were taking finasteride as part of their treatment. We compared the therapy results to those of a placebo and looked at the data before and after the therapy. The primary outcome was the alteration in prostate-specific antigen (PSA) levels assessed across follow-up periods ranging from several months to multiple years. Search queries incorporated Medical Subject Headings (MeSH) and relevant keywords, including: (benign prostatic hyperplasia[MeSH Terms]) OR (BPH[Title/Abstract]) OR (benign prostate enlargement[Title/Abstract]) AND (finasteride[MeSH Terms]) OR (5-alpha reductase inhibitor[Title/Abstract]) AND (prostate specific antigen[MeSH Terms]) OR

(Serum PSA[Title/Abstract]) OR (PSA level[Title/Abstract]). Eligible studies were observational in design, written in English, conducted on human participants, and had no restrictions on study length or year of publication.

The eligibility criteria mandated that (a) all participants be exclusively diagnosed with BPH, excluding prostate cancer; (b) the study examines the administration of finasteride in relation to fluctuations in PSA levels; and (c) the research design includes randomized controlled trials (RCTs) or observational formats, such as cohort or case-control studies. Two reviewers collected data independently, utilizing Rayyan.ai for the preliminary screening and selection process. We saved files from PubMed and ScienceDirect in the RIS and BibTeX formats. Reading the titles and abstracts was the first step in the evaluation process. After then, every article that met the first set of criteria was read all the way through.

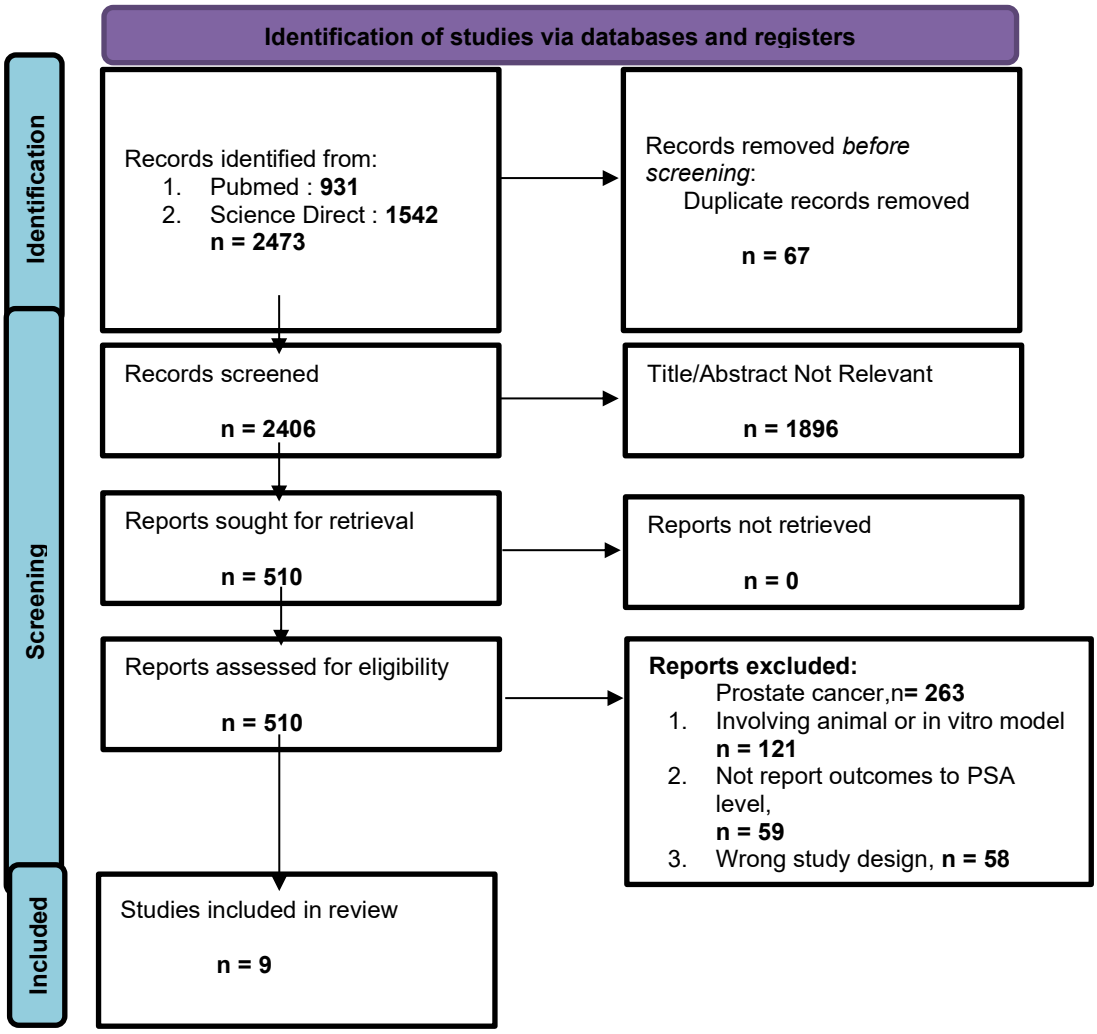

Figure 1. Prisma Flow Chart

RESULT AND DISCUSSION

The initial search of the selected databases yielded 2,473 potentially helpful publications. After removing duplicate entries and completing an initial examination of the titles and abstracts, numerous studies were left out because they didn't have enough data to show how

finasteride affected men's blood PSA levels or weren't related to the study question. A thorough full-text assessment of the remaining papers, based on the predetermined inclusion and exclusion criteria, further streamlined the list. Table 1 shows that only nine of the papers satisfied all of the requirements and were included in the final analysis of this systematic review.

Table 1. Presentation of the studies selected

No	Author, Year	Mean Age	Sample Size	Follow Up	Total PSA	Free PSA	Prostate Volume	PSAD
1	Matzkin, 1996	71 years	20	9 months	3.57 --> 1.99	0.60 --> 0.35	-	-
2	Keetch, 1997	69 years 8 months	69	6 months	2.8 --> 1.1	-	-	-
3	Lee, 2007	71 years 7 months	44	6 months	6.74 --> 3.83	2.57 --> 1.45	56.0 --> 54.9	0.15 --> 0.09
4	Chiu, 2004	72 years 5 months	166	6 months	2.48 --> 1.57	-	39.83 --> 33.62	0.077 --> 0.055
5	Pannek, 1998	64 years 6 months	41	6 months	3.02 --> 1.49	0.39 --> 0.20	36.7 --> 29.6	0.077 --> 0.046
6	Kaplan, 2002	60 years 6 months	38	12 months	6.32 --> 3.73	-	37.3 --> 30.4	-
7	Handel, 2006	67 years 2 months	23	6 months	9.34 --> 5.09	-	-	-
8	Chiu, 2023	-	164	12 months	8.9 --> 4.4	1.6 --> 0.8	-	-
9	Stoner, 1994	64 years & 66 years	1098	36 months	2.4 --> 1.2 & 4.0 --> 2.0	-	58.5 --> 43 & 48.8 --> 35	-

The synthesis of existing evidence demonstrates that finasteride therapy in men diagnosed with benign prostatic hyperplasia (BPH) consistently produces measurable changes in various clinical and biochemical markers, particularly total PSA, free PSA, prostate volume, and prostate-specific antigen density (PSAD). The magnitude of decline varied among studies; nevertheless, the general trend remained stable, aligning with finasteride's established pharmacological role as a 5-α reductase inhibitor. Total PSA showed the most consistent and substantial reductions across all levels, with drops of 30% to over 50% from the baseline across 6 to 12 months. Over nine months, Matzkin's report showed a drop from 3.57 to 1.99 ng/mL, Keetch's report showed a drop from 2.8 to 1.1 ng/mL, and Lee's report showed a drop from 6.74 to 3.83 ng/mL (. Chiu, Pannek, Kaplan, Handel, and Stoner have done other studies that show similar trends, with PSA levels going down in different groups of patients. These consistent results underscore the need of utilizing correction factors when evaluating PSA values for prostate cancer screening in persons undergoing finasteride therapy.

Data on free PSA were provided less often but generally demonstrated a falling trend similar to total PSA. Matzkin's level reduced from 0.60 to 0.35 ng/mL over one year; Lee's from 2.57 to 1.45 ng/mL; Pannek's from 0.39 to 0.20 ng/mL; and Chiu's from 1.6 to 0.8 ng/mL (P. K. F. Chiu et al., 2023). The levels of free PSA and total PSA may have gone down at the same rate, but it doesn't mean they went down in the same way. This might change the ratio of free PSA to total PSA. patients typically use this ratio to discern the difference between BPH and prostate cancer, thus these alterations might influence how patients who use finasteride are checked.

Tests indicated that the prostate volume fell reduced, generally by small but regular amounts. Lee's volume went down from 56.0 to 54.9 cm^3, Kun Yuan's from 39.83 to 33.62 cm^3, Pannek's from 36.7 to 29.6 cm^3, and Kaplan's from 37.3 to 30.4 cm^3. Stoner observed more pronounced losses, with values declining from 58.5 to 43 cm^3 and from 48.8 to 35 cm^3 over two separate cohorts (P. K. F. Chiu et al., 2023). These drops are in accordance with finasteride's propensity to lower androgens, which causes epithelial cells to die off and the prostate to shrink a bit. The concurrent change in PSA and volume supports the concept that decreasing PSA levels signify a reduction in tissue size.

Some studies that looked at variations in PSAD showed a strong connection between PSA levels and prostate growth. Lee's value went down from 0.15 to 0.09 $ng/mL/cm^3$, Kun Yuan's from 0.077 to 0.055, Pannek's from 0.077 to 0.046, and Handel's from 0.20 to 0.12.

In several cases, the PSA level went down more than the volume, which caused the PSAD readings to go down. If the important clinical modifications aren't achieved, these types of alterations might make it tougher to diagnose cancer. This is because PSAD is often used to assist figure out how likely it is that someone has prostate cancer.

CONCLUSION

Finasteride treatment consistently reduces total PSA levels by 30% to over 50% within 6 to 12 months, a finding observed across diverse study populations and designs, primarily through the inhibition of intraprostatic dihydrotestosterone synthesis. While reductions in free PSA are also noted, they are generally smaller than those in total PSA. Prostate size decreases by approximately 5% to 20%, with corresponding drops in PSA density, highlighting the influence of prostate volume changes on serum PSA. These results reinforce finasteride’s dual benefits of lowering PSA and shrinking the prostate, thereby improving BPH management and refining prostate cancer screening protocols through appropriate PSA adjustment factors. Future research should focus on long-term effects on free PSA, optimal adjustment models for PSA interpretation during finasteride therapy, and the drug’s role in reducing prostate cancer incidence without masking high-risk cases.

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