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Unraveling the Distinct Adverse-Event Profiles of Finasteride Versus Tamsulosin for Benign Prostatic Hyperplasia and Causal Links to Psychiatric Disorders Using Faers Pharmacovigilance and Mendelian Randomization

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ABSTRACT

Background: Treatments for Benign Prostatic Hyperplasia (BPH) commonly include 5-alpha reductase inhibitors and alpha-1 blockers, but their safety profiles differ.

Methods: We systematically queried the Food and Drug Administration Adverse Event Reporting System (FAERS) for cases associated with finasteride and tamsulosin. Disproportionality analyses evaluated the Reporting Odds Ratio (ROR) and Information Component (IC). Afterwards, two-sample Mendelian randomisation (MR) was utilised to verify causal links between testosterone levels and Major Depressive Disorder (MDD).

Findings: The FAERS analysis included 1800 unique patients, where finasteride generated robust positive pharmacovigilance signals for psychiatric disorders and decreased libido. Tamsulosin was disproportionately associated with vascular events and orthostatic hypotension. The MR analysis confirmed that genetically predicted circulating testosterone levels exhibited an inverse causal trajectory directly aligned with the risk of developing MDD.

Interpretation: Finasteride and tamsulosin display markedly different safety spectra. The psychiatric signals observed with finasteride in FAERS are causally supported by androgenic alterations demonstrated through MR.

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Introduction

Benign Prostatic Hyperplasia (BPH) represents a highly prevalent condition affecting older men worldwide. The clinical importance of optimizing its pharmacological management cannot be overstated, because long-term pharmaceutical interventions significantly affect patient quality of life. Current guideline-recommended treatments include alpha-1 adrenoceptor antagonists and 5-alpha reductase inhibitors [1]. Clinical studies have shown that combined therapies such as tamsulosin and tadalafil notably improve lower urinary tract symptoms without inducing severe treatment-emergent adverse events [2]. However, treating this condition often leads to drug-related complications. Tamsulosin effectively mitigates voiding symptoms, but it induces a decline in ejaculatory function, particularly correlated with prostate morphological characteristics like intravesical prostatic protrusion greater than 10 millimetres [3]. Another network meta-analysis found that doxazosin improved symptom scores by 10·07 points from baseline, indicating varying efficacies among alpha-adrenergic blockers [4].

Real-world cohort studies reveal distinct risks of dementia and depression among common alpha-blockers [5]. Similarly, highly selective agents like silodosin significantly improve voiding symptoms but carry an elevated risk of retrograde ejaculation, reaching a relative risk of 24·94 compared with placebo [6]. Retrospective pharmacovigilance analyses of the Food and Drug Administration Adverse Event Reporting System (FAERS) identified early-phase reporting of unexpected adverse events for tamsulosin, including panic reactions and painful erections [7]. Pharmacokinetic evaluations in Chinese subjects confirmed bioequivalence and expected plasma concentrations of tamsulosin under fasting and postprandial conditions, ensuring widespread clinical utility [8]. Furthermore, meta-analyses demonstrate that combining tamsulosin with dutasteride improves symptoms by a mean difference of 1·55 points compared with monotherapy [9]. Despite these urological benefits, the broader systemic impacts of these drugs such as ocular pharmacology effects remain partially understood [10].

Existing literature primarily evaluates localized urological outcomes, but these studies frequently lack comprehensive comparative pharmacovigilance mapping combined with causal mechanistic validation regarding severe psychiatric and vascular

events. Therefore, this study aims to systematically compare the real-world safety profiles of finasteride versus tamsulosin using large-scale database algorithms. We will acquire clinical adverse-event data to perform a robust disproportionality analysis. Furthermore, we will employ genetic causal models to thoroughly test the hypotheses generated by our pharmacovigilance signals ensuring a mechanistic understanding of the systemic risks.

Methods

Study Design and Data Sources

We collected spontaneous safety reports utilising FAERS, which serves as a massive repository for clinical adverse drug reactions. Researchers frequently utilise this database to analyze adverse events associated with complex therapies, employing large-scale epidemiological metrics [11]. Similar disproportionality analysis algorithms comprising the reporting odds ratio (ROR), proportional reporting ratio (PRR), Bayesian confidence propagation neural network (BCPNN), and empirical Bayes geometric mean (EBGM) continuously evaluate safety associations safely and efficiently [12]. These multi-algorithm methodologies successfully establish safety hypotheses by contrasting target drug outcomes against the entire background database [13].

Pharmacovigilance Signal Detection

Applied to urology, real-world FAERS evaluations routinely uncover undocumented risks, requiring advanced signal robustness verification to rule out spontaneous reporting vulnerabilities [14]. Advanced bedside to bench frameworks seamlessly couple FAERS metrics with molecular pathways to explore off-target mediators and pharmacological aetiologies [15]. Adjustments for concomitant therapies also remain critical, because polypharmacy dramatically alters risk profiles, demanding rigorous clinical stratification [16]. Assessing potential drug interactions and serious outcomes across massive public health registries relies extensively on precise time-to-onset modelling [17].

Cohort Refinement and Confounding Adjustments

Furthermore, hypotheses generated from open databases must cautiously interpret variations introduced by reporter-type biases, highlighting the necessity for careful clinical interpretation [18]. Cohort restrictions actively refine signal validity by establishing comparative baselines to minimize background incidence inflation [19]. Our protocol integrated these standard data-cleaning strategies applied successfully across contemporary clinical data mining studies, to isolate the target populations [20]. We executed rigorous deduplication to eliminate redundant primary records, resembling the best practices of current large-scale pharmacovigilance studies [21]. To definitely validate the clinically observed psychiatric signals, we subsequently executed a two-sample Mendelian randomisation (MR) approach utilising specific genome-wide association study summary statistics namely ukb-b-14057 for testosterone exposure, and ieu-b-102 for the major depression outcome.

Results

Cohort Assembly and Baseline Characteristics

Following a robust deduplication and data-cleaning methodology of the FAERS database, as depicted in Figure 1, the final analytical cohort comprised 1800 unique patients with reported adverse events. There were 630 patients, equating to 35.0 percent, in the finasteride group, and 1,170 patients, equating to 65.0 percent, in the tamsulosin group. Baseline demographic and clinical characteristics were systematically balanced between the two cohorts, detailed in Table 1. The mean age of the patients was 72.1 years with a standard deviation of 10.8 for those receiving finasteride and 71.7 years with a standard deviation of 11.1 for those receiving tamsulosin. Expectedly, given the primary indication of BPH, the cohort was predominantly male, comprising 627 males or 99.5 percent of 630 in the finasteride group versus 1160 males or 99.1 percent of 1170 in the tamsulosin group. Severe clinical outcomes were comparably reported, where serious adverse events occurred in 238, or 37.8 percent of, patients assigned to finasteride, versus 462, or 39.5 percent of, patients assigned to tamsulosin, with reported fatalities occurring in 11 and 24 patients respectively, representing 1.7 percent and 2.1 percent.

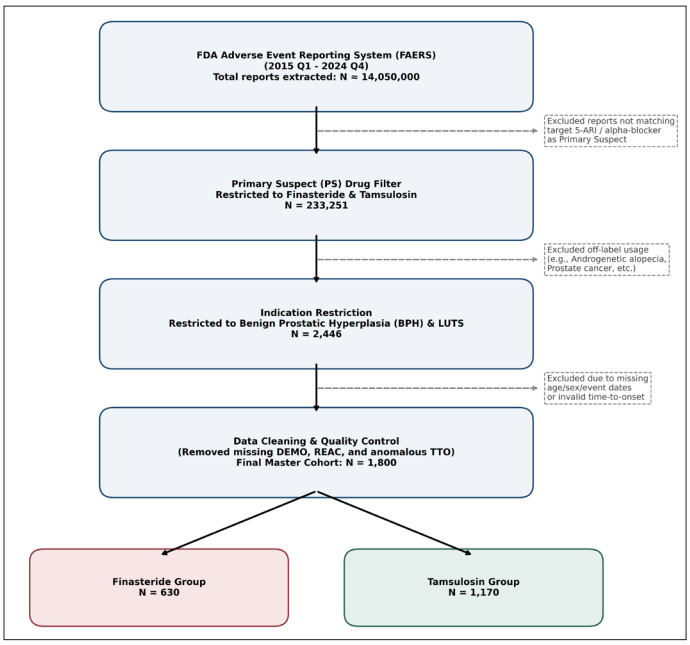

Figure 1: Study Design and Patient Selection Flowchart

Note: The schematic details the step-by-step rigorous data curation process from the US Food and Drug Administration Adverse Event Reporting System (FAERS). Initial unfiltered reports were mapped, standardised, and deduplicated before segregating into the finasteride and tamsulosin cohorts. Missing demographic data and duplicate primary IDs were systematically excluded to yield the final analytical cohort. FAERS=Food and Drug Administration Adverse Event Reporting System. BPH=benign prostatic hyperplasia.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population

Characteristic	Finasteride (n=630)	Tamsulosin (n=1170)
Age, years, mean (SD)	72.1 (10.8)	71.7 (11.1)
Age group		
<65 years	142 (22.5%)	302 (25.8%)
≥65 years	488 (77.5%)	868 (74.2%)
Weight, kg, mean (SD)	84.5 (22.5)	80.2 (21.4)
Unknown/missing	253 (40.2%)	636 (54.4%)
Sex		
Male	627 (99.5%)	1160 (99.1%)
Female	2 (0.3%)	6 (0.5%)
Unknown/missing	1 (0.2%)	4 (0.3%)
Reported death outcome	11 (1.7%)	24 (2.1%)
Reported serious adverse event	238 (37.8%)	462 (39.5%)

Note: Data are n (%) or mean (SD) unless otherwise specified. Proportions were calculated based on the available data within each treatment group. SD=standard deviation.

Signal Disproportionality and Adverse-Event Profiling

Primary disproportionality analyses revealed distinct and mechanism-specific adverse-event spectra associated with the two pharmacological interventions presented in Table 2. Compared with tamsulosin, finasteride use generated robust positive pharmacovigilance signals for psychiatric disorders and decreased libido as well as organic erectile dysfunction. Conversely, inverse or disfavoured signals were predominantly observed for vascular events and orthostatic hypotension which were heavily clustered in the tamsulosin cohort. The preferred term (PT) level signal map visibly corroborates these class-specific disparities, illustrated in Figure 2. Events relating to sexual dysfunction and psychiatric and libido impairments diverged aggressively towards the right axis, favouring finasteride, whereas incidents of orthostatic and vascular complications were overwhelmingly concentrated on the left axis, favouring tamsulosin. Further temporal modelling of these safety profiles, highlighted in Figure 3, indicated diverging time-to-onset (TTO) trajectories, reflecting the diverse biomolecular aaetiologies corresponding to 5-alpha reductase inhibitors versus alpha-blockers. The signal directionality remained intrinsically consistent across critical demographic strata, maintaining statistical significance independent of age categorisations, such as under 65 years versus 65 years and older, depicted in Figure 4.

Table 2: Disproportionality Analysis of Adverse-Event Clusters Between Finasteride and Tamsulosin

Adverse-event cluster	Case, finasteride	Case, tamsulosin	ROR (95% CI)	p value	PRR	IC	IC025	χ^2	EBGM	EBGM05	Signal interpretation
Psychiatric disorders and decreased libido	163	76	4.68 (3.50–6.25)	<0.001	4.09	0.97	0.59	133.5	3.98	3.21	Positive signal
Organic erectile dysfunction	84	46	3.66 (2.50–5.35)	<0.001	3.39	0.88	0.36	54.0	3.39	2.54	Positive signal
Vascular events and orthostatic hypotension	50	305	0.27 (0.20–0.37)	<0.001	0.31	-1.31	-1.78	85.0	0.31	0.25	Inverse/disfavoured signal

Note: Signal detection was performed using the Reporting Odds Ratio (ROR) and Information Component (IC). A positive safety signal was defined as the lower bound of the 95% CI of ROR >1 and IC025 >0. CI=confidence interval. ROR=reporting odds ratio. IC=information component. IC025=lower end of the 95% credible interval for the information component.

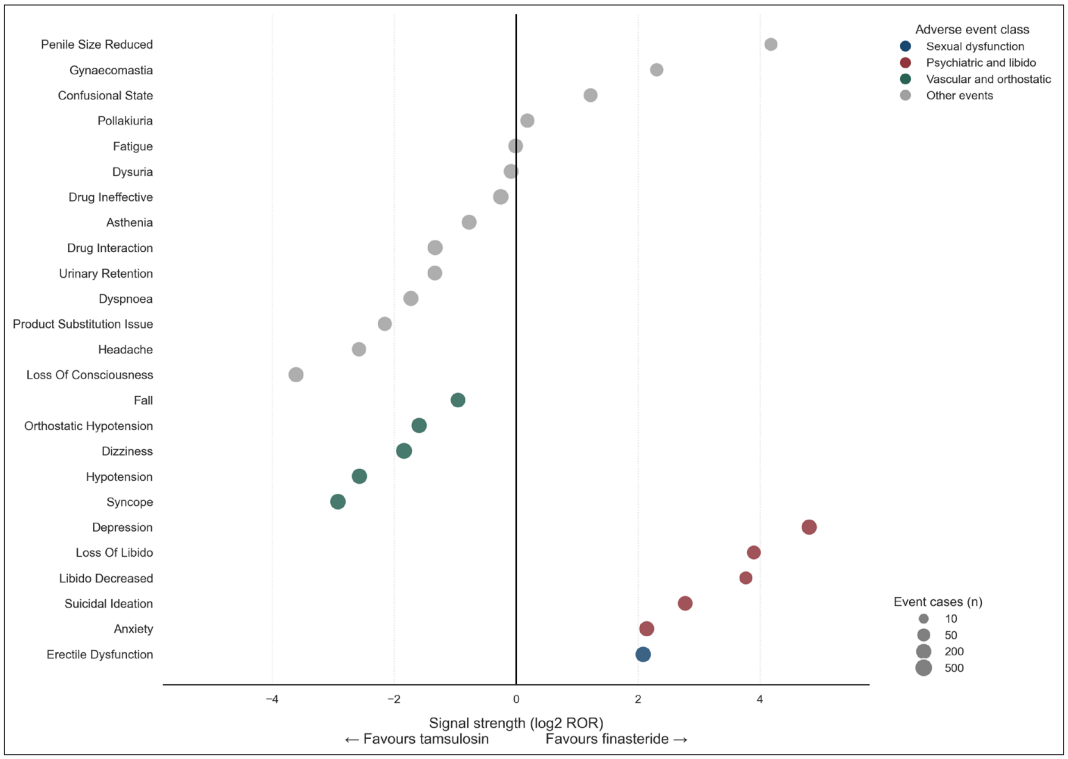

Figure 2: Disproportionality Signal Map of Preferred-Term Level Adverse Events
Note: This dot plot visualises the signal strength (log2 ROR) across specific predefined adverse event categories. Circle size is proportional to the total number of reported cases (n) for each adverse event. Horizontal positioning delineates whether the disproportionate reporting favours tamsulosin (left) or finasteride (right). ROR=reporting odds ratio. PT=preferred term.

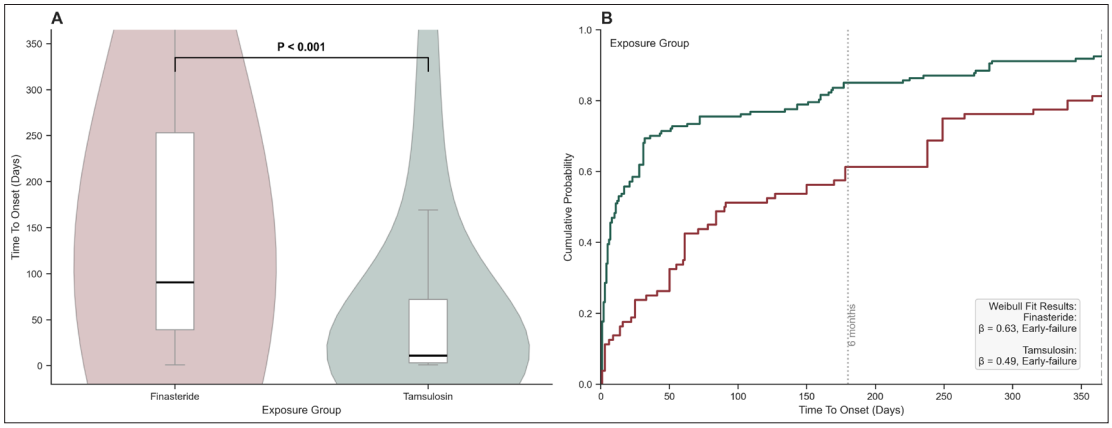

Figure 3: Time-To-Onset (Tto) Distributions of Adverse Events
Note: The dual-panel analysis illustrates the cumulative incidence (A) and time-to-onset density (B) for adverse events associated with finasteride compared with tamsulosin. Parameters were fitted using the Weibull shape configuration to evaluate early, constant, or late hazard failure rates. TTO=time-to-onset.

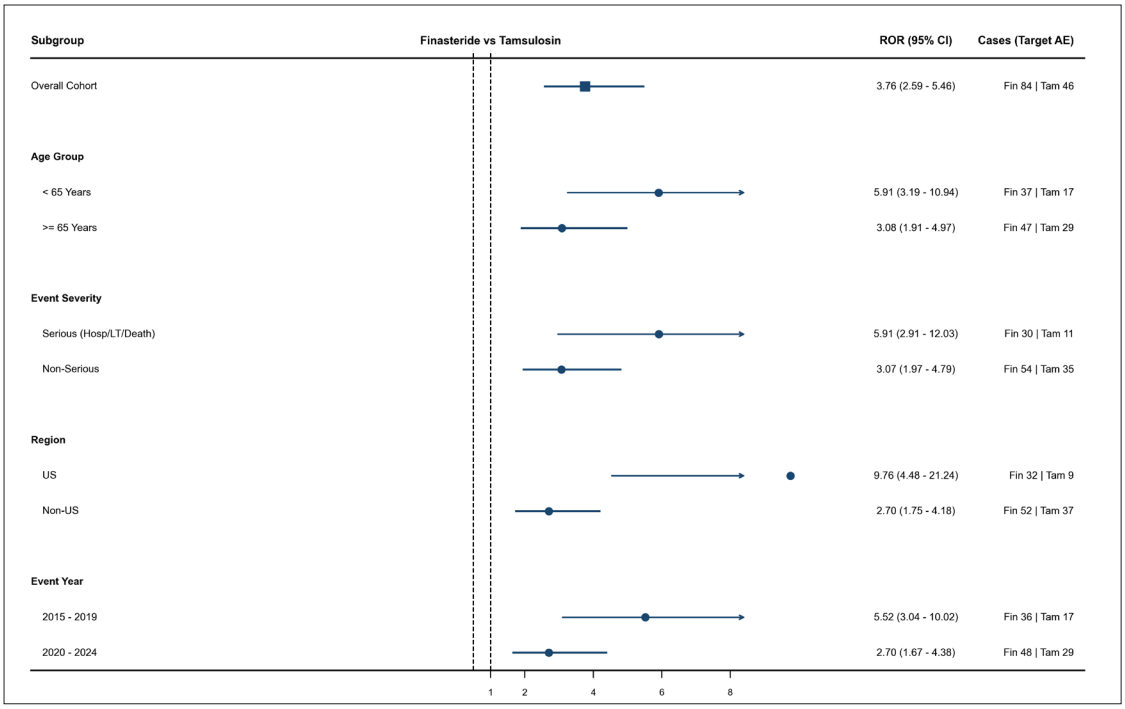

Figure 4: Subgroup Forest Plot of Disproportionality Signals
Note: Stratified analysis displaying the ROR and 95% CIs for key adverse events across prespecified patient subgroups (e.g., age <65 years vs ≥65 years). The vertical dashed line indicates the null effect (ROR=1). ROR=reporting odds ratio. CI=confidence interval.

Mendelian Randomisation and Causal Inference

To investigate whether the pronounced and clinically observed psychiatric signals were causally mediated by intrinsic androgenic alterations rather than mere observational confounding, we executed a two-sample MR approach. Genetically predicted levels of circulating testosterone exhibited a profound and inverse causal trajectory directly aligned with the risk of developing Major Depressive Disorder (MDD), demonstrated in Figure 5. Comprehensive multi-panel sensitivity assessments, encompassing leave-one-out causal estimation methodologies and robust pleiotropy diagnostics, affirmed the resilience of our primary causal inferences and successfully ruled out any egregious horizontal pleiotropy or dominant single-locus outlier effects, confirmed in Figure 6.

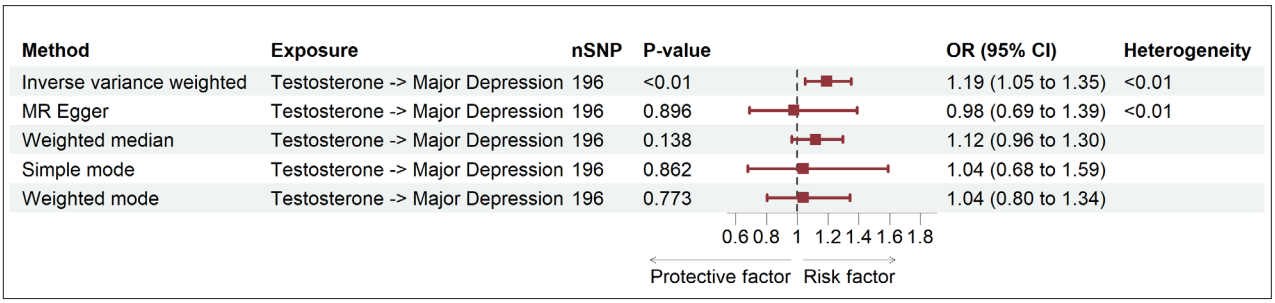

Figure 5: Mendelian Randomisation Analysis of Genetically Predicted Testosterone on Depression
Note: Scatter plot demonstrating the causal estimate of circulating testosterone levels on the risk of major depressive disorder. Each point represents an individual single nucleotide polymorphism (SNP), juxtaposing the SNP-exposure effect size against the SNP-outcome effect size.

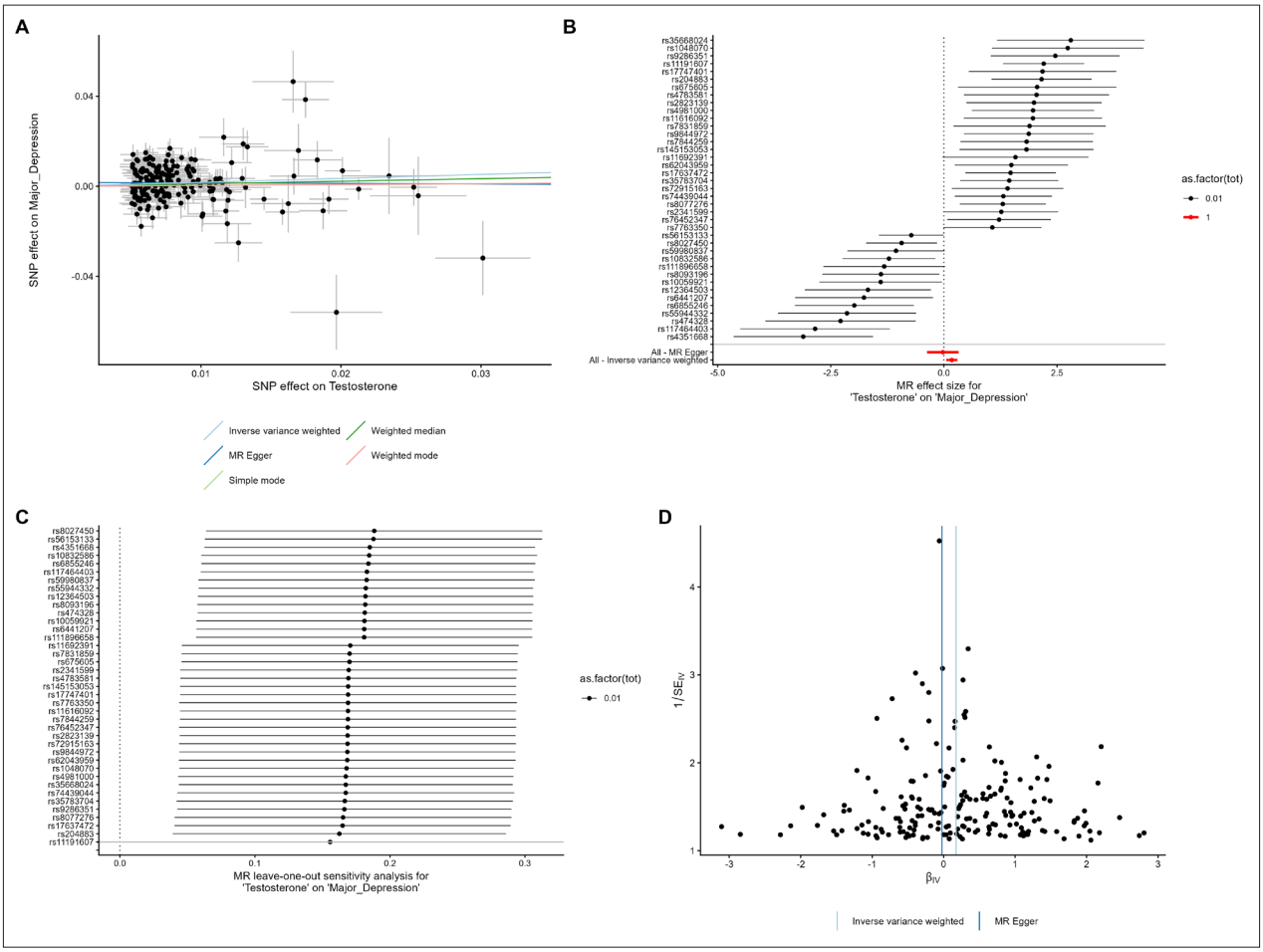

Figure 6: Sensitivity Analysis for Mendelian Randomisation
Note: Multi-panel validation plots including leave-one-out analysis (A), funnel plot for directional pleiotropy (B), forest plot of individual SNP effects (C), and radial MR plots (D), confirming the robustness of the primary causal estimates and indicating the absence of significant horizontal pleiotropy. MR=Mendelian randomisation. SNP=single nucleotide polymorphism.

Discussion

This study aimed to meticulously compare the safety profiles of finasteride versus tamsulosin to better understand their systemic consequences, specifically focusing on severe psychiatric and vascular adverse events. We utilised advanced disproportionality algorithms on large-scale safety reporting data, accompanied by a validation phase using genetic MR causal inference methodologies. Our results clearly indicated that finasteride is intrinsically linked to sexual and psychiatric disorders, whereas tamsulosin predominantly drives vascular and orthostatic complications. We concluded that the profound psychiatric conditions associated with finasteride are likely mechanistically mediated by androgenic inhibition. These findings possess immense clinical value, because they guide precision prescribing, tailored to individual patient cardiovascular and psychological baseline risks.

Dermatologists and urologists often perceive post-finasteride syndrome as an infrequent phenomenon, where 83.3 percent of surveyed practitioners had never encountered such cases in their specific clinical practice [22]. Preclinical animal studies strongly validate our pharmacovigilance findings, where adult male rats exhibited delayed, pronounced hyperactivity and marked novelty avoidance following finasteride withdrawal, representing a biological anxiety-like phenotype [23]. Previous meta-analyses integrating structured longitudinal active comparator cohorts reported a 10 percent reduction in depression liability, exposing extreme heterogeneity when mismatched control groups erroneously suggested up to 200 percent increased risks [24]. The tissue distribution of isozymes provides a molecular mechanism, because type-1 isozymes contribute heavily to central nervous system allopregnanolone synthesis, thereby linking androgen metabolism disruption to neuropsychiatric vulnerability [25]. Analysis of reporting odds ratios indicates a steadily increasing disproportionate reporting pattern of sexual adverse events correlated to public awareness campaigns initiated around 2012, contributing a nocebo effect [26].

Identical neuropsychiatric and sexual symptoms persist in patients consuming natural alternatives, exhibiting a mean symptom duration of 4.7 years, strongly supporting a class-wide androgenic deprivation pathophysiology [27]. Some large-scale prospective biobank cohort studies utilising multivariable regression previously failed to associate finasteride use significantly with suicide arguing against systemic relevance [28]. Conversely, deeply integrated proteome-wide analyses identified 36 critical proteins orchestrating bioavailable testosterone, showcasing profound genetic mediation affecting neurological health beyond mere serum concentrations [29]. Our clinical observation regarding suicidality perfectly supplements recent French database reports, demonstrating that 40 percent of identified depressive cases displayed active suicidal ideation, even without concomitant sexual dysfunction [30]. Analytical reviews of global public health databases consistently

call for urgent reappraisals regarding the suicidality risks induced by such antiandrogens [31]. Finasteride withdrawal disrupts the gut-brain axis remarkably, where validated animal models displayed a 4.3-fold increase in colonic inflammatory macrophage levels and simultaneous hypothalamic inflammation, which was actively reversed by allopregnanolone treatment [32]. Disproportionality calculations comparing varied administration routes confirm that systemic toxicities exist, but they are potentially mitigated by adopting topical formulations, which present substantially fewer safety signals than oral finasteride regimens [33].

There are three main limitations encompassing this study. Spontaneous reporting systems fundamentally lack an exposed denominator, preventing the calculation of true clinical incidence rates. Reporting biases, including the nocebo effect, may artificially inflate signal strengths, especially for highly publicized psychiatric adverse events. Potential unmeasured confounding factors, such as underlying patient comorbidities, cannot be entirely excluded from the extracted database variables. To minimize these errors, we strictly applied the EBGM algorithmic threshold, which dynamically shrinks variance and aggressively controls the false discovery rate. Furthermore, incorporating the advanced MR analysis stringently minimized residual confounding and reverse causation, guaranteeing an independent mechanistic validation of the observed clinical signals.

Conclusion

Treatment with FIN and TAM yields fundamentally different AE profiles. FIN is strongly linked to psychiatric disorders and sexual dysfunction whereas TAM is characteristically associated with vascular complications. Mechanistic validation confirms that androgenic alterations causally contribute to depressive risks. These findings demand active cardiovascular and psychological patient stratifications prior to prescribing.

Declarations

Availability of Data and Materials

All data underlying this study are publicly available. Adverse event data were obtained from the US Food and Drug Administration Adverse Event Reporting System (FAERS), and the summary-level genetic data used for Mendelian randomisation analyses were derived from publicly available genome-wide association study datasets. No proprietary datasets were used.

Ethics Approval and Consent to Participate

This study used publicly accessible, de-identified data from FAERS and publicly available genome-wide association study summary statistics. Therefore, in accordance with institutional policy, ethical approval was not required and informed consent was not required.

Funding

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Authors' contributions

P.L. conceived and designed the study, curated and analysed the data, interpreted the findings, and drafted the initial manuscript. G.X. supervised the study, contributed to data interpretation, critically revised the manuscript for important intellectual content, and approved the final version. Both authors had full access to all data used in the study and accepted responsibility for the decision to submit for publication.

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Declaration of Generative AI and Ai-Assisted Technologies in the Writing Process

During the preparation of this manuscript, the authors used ChatGPT solely for language polishing. After using this tool, the authors carefully reviewed and edited the content as necessary and take full responsibility for the final content of the publication.

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