

RESEARCH MONOGRAPH . COMPOUND PROFILE

# Finasteride

*A 4-azasteroid 5-alpha-reductase type II inhibitor; approved for benign prostatic hyperplasia and androgenetic alopecia.*

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## ABSTRACT

Finasteride (N-(2-methyl-2-propenyl)-3-oxo-(5- $\alpha$ ,17- $\beta$ )-4-azaandrost-1-ene-17-carboxamide; CAS 98319-26-7; molecular formula C<sub>23</sub>H<sub>36</sub>N<sub>2</sub>O<sub>2</sub>; molecular weight 372.55) is a 4-azasteroid 5- $\alpha$ -reductase inhibitor developed at Merck and approved by the FDA in 1992 (Proscar 5 mg for BPH) and 1997 (Propecia 1 mg for androgenetic alopecia). Mechanism: selective inhibition of 5- $\alpha$ -reductase type II isozyme (K<sub>i</sub> approximately 10 nM), which converts testosterone to dihydrotestosterone (DHT) in prostate, hair follicle, and skin. Plasma DHT is reduced by approximately 65 to 70 percent at the 5 mg dose. Type I 5AR (in liver, skin, and brain) is largely unaffected. Plasma half-life is approximately 6 to 8 hours; metabolism is via CYP3A4. The compound has well-documented sexual side effects (reduced libido, erectile dysfunction in approximately 1 to 5 percent of users) and the controversial post-finasteride syndrome (persistent symptoms after discontinuation, debated as a clinical entity). Used as the reference 5AR-II selective inhibitor in mechanism studies and as a positive control in androgen pathway research.

# 1. Compound Identification

**Finasteride** is described as 5-alpha-reductase type ii inhibitor. A 4-azasteroid 5-alpha-reductase type II inhibitor; approved for benign prostatic hyperplasia and androgenetic alopecia.

## 1.1 Class summary

5-alpha-reductase type II inhibitor

## 1.2 Subtitle

A 4-azasteroid 5-alpha-reductase type II inhibitor; approved for benign prostatic hyperplasia and androgenetic alopecia.

## 2. Abstract

Finasteride (N-(2-methyl-2-propanyl)-3-oxo-(5- $\alpha$ ,17- $\beta$ )-4-azaandrost-1-ene-17-carboxamide; CAS 98319-26-7; molecular formula C<sub>23</sub>H<sub>36</sub>N<sub>2</sub>O<sub>2</sub>; molecular weight 372.55) is a 4-azasteroid 5- $\alpha$ -reductase inhibitor developed at Merck and approved by the FDA in 1992 (Proscar 5 mg for BPH) and 1997 (Propecia 1 mg for androgenetic alopecia). Mechanism: selective inhibition of 5- $\alpha$ -reductase type II isozyme (K<sub>i</sub> approximately 10 nM), which converts testosterone to dihydrotestosterone (DHT) in prostate, hair follicle, and skin. Plasma DHT is reduced by approximately 65 to 70 percent at the 5 mg dose. Type I 5AR (in liver, skin, and brain) is largely unaffected. Plasma half-life is approximately 6 to 8 hours; metabolism is via CYP3A4. The compound has well-documented sexual side effects (reduced libido, erectile dysfunction in approximately 1 to 5 percent of users) and the controversial post-finasteride syndrome (persistent symptoms after discontinuation, debated as a clinical entity). Used as the reference 5AR-II selective inhibitor in mechanism studies and as a positive control in androgen pathway research.

### 3. Mechanism of Action

Selective 5-alpha-reductase type II inhibition; reduces conversion of testosterone to DHT. Largely spares type I 5AR.

The mechanistic profile described above represents the consensus position from peer-reviewed literature where available; for compounds with sparse formal characterization, this section reflects vendor-grade reports and structurally-related class pharmacology and should be interpreted with appropriate caution.

## 4. Pharmacokinetics and Routes of Administration

Pharmacokinetic data are summarized within the abstract for compounds with formal Phase 1 records. Compounds without published human pharmacokinetic studies are noted as such; investigators should treat the absence of data as a research limitation.

## 5. Reported Research Dose Ranges

Clinical 1 mg (alopecia) or 5 mg (BPH) per oral administration daily. Rodent studies 0.5 to 5 mg/kg/day.

Doses are scaled from clinical use where the compound is approved as a medicine, from Phase 2/3 trials where development advanced, or from rodent dose-response data scaled by allometric assumptions where the compound has no human clinical record. Investigators should treat dose ranges from research-chemical-grade vendors as starting points for their own pharmacokinetic and tolerability characterization rather than validated dosing recommendations.

## 6. Sourcing and Quality Verification

Investigators should obtain analytical confirmation of identity (mass spectrometry; for peptides, additionally amino acid analysis or N-terminal sequencing) and purity (HPLC area-percent, ideally greater than 95 percent) on every lot. Vendor certificates of analysis vary in quality; third-party analytical confirmation through independent laboratories (e.g., Janoshik Analytical, Anabolic Lab) is the gold standard for research-grade peptides and small molecules.

For compounds with established pharmaceutical-grade marketing in any jurisdiction, sourcing from the marketed product (where legal and accessible) provides the highest quality assurance.

## 7. Reconstitution and Handling

Peptides should be reconstituted in bacteriostatic water or sterile water for injection in the volume appropriate to the desired concentration; lyophilized peptides should be allowed to come to room temperature before reconstitution to minimize precipitation. Reconstituted peptides should be stored refrigerated and used within the timeframe specified by the manufacturer (typically 14 to 30 days for bacteriostatic water reconstitutions, shorter for sterile water).

Small molecules: storage conditions vary by compound; consult the certificate of analysis for the specific product. Most are stable at room temperature in dry, dark conditions; oxidation-sensitive compounds (e.g., catecholamine precursors) require cool storage and protection from light.

## 8. Stack Interaction Considerations

Investigators combining this compound with other research substances should consider receptor-level interactions (additive effects at shared targets), enzyme-level interactions (CYP-mediated drug-drug interactions where applicable), and pharmacodynamic interactions (additive sedation, additive serotonergic activity, additive hemodynamic effects).

The stack interaction section in published monographs of well-characterized compounds is necessarily incomplete for any specific combination; investigators should treat novel combinations with elevated caution and titrate gradually.

## 9. References

1. Steiner JF. Clinical pharmacokinetics and pharmacodynamics of finasteride. Clin Pharmacokinet 1996.
2. Kaufman KD, et al. Finasteride in the treatment of men with androgenetic alopecia. J Am Acad Dermatol 1998.
3. Trost L, et al. The recently published Cochrane review of post-finasteride syndrome. Sex Med Rev 2018.

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