

RESEARCH MONOGRAPH . COMPOUND PROFILE

Dutasteride

A 4-azasteroid dual 5-alpha-reductase inhibitor (types I and II); more complete DHT suppression than finasteride.

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COMPOUND CLASS	Dual 5-alpha-reductase I/II inhibitor

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ABSTRACT

Dutasteride ((5- α ,17- β)-N-{2,5-bis(trifluoromethyl)phenyl}-3-oxo-4-azaandrost-1-ene-17-carboxamide; CAS 164656-23-9; molecular formula C₂₇H₃₀F₆N₂O₂; molecular weight 528.53) is a 4-azasteroid dual 5- α -reductase inhibitor developed at GlaxoSmithKline and approved by the FDA in 2002 under the trade name Avodart. Distinct from finasteride by inhibition of both 5AR isozymes (type I Ki approximately 0.6 nM, type II Ki approximately 0.3 nM, both substantially more potent than finasteride at type II). Plasma DHT is reduced by approximately 90 to 95 percent at the 0.5 mg dose, compared to 65 to 70 percent for finasteride. Plasma half-life is approximately 5 weeks (the longest in the 5AR inhibitor class), reflecting tight tissue binding. Approved for benign prostatic hyperplasia; off-label use in androgenetic alopecia (often more effective than finasteride). The longer half-life means slower offset of effect on discontinuation but also slower washout for fertility planning. Sexual side effects parallel finasteride. Used as the canonical dual 5AR inhibitor in mechanism studies.

1. Compound Identification

Dutasteride is described as dual 5-alpha-reductase i/ii inhibitor. A 4-azasteroid dual 5-alpha-reductase inhibitor (types I and II); more complete DHT suppression than finasteride.

1.1 Class summary

Dual 5-alpha-reductase I/II inhibitor

1.2 Subtitle

A 4-azasteroid dual 5-alpha-reductase inhibitor (types I and II); more complete DHT suppression than finasteride.

2. Abstract

Dutasteride ((5- α ,17- β)-N-{2,5-bis(trifluoromethyl)phenyl}-3-oxo-4-azaandro-1-ene-17-carboxamide; CAS 164656-23-9; molecular formula C₂₇H₃₀F₆N₂O₂; molecular weight 528.53) is a 4-azasteroid dual 5- α -reductase inhibitor developed at GlaxoSmithKline and approved by the FDA in 2002 under the trade name Avodart. Distinct from finasteride by inhibition of both 5AR isozymes (type I Ki approximately 0.6 nM, type II Ki approximately 0.3 nM, both substantially more potent than finasteride at type II). Plasma DHT is reduced by approximately 90 to 95 percent at the 0.5 mg dose, compared to 65 to 70 percent for finasteride. Plasma half-life is approximately 5 weeks (the longest in the 5AR inhibitor class), reflecting tight tissue binding. Approved for benign prostatic hyperplasia; off-label use in androgenetic alopecia (often more effective than finasteride). The longer half-life means slower offset of effect on discontinuation but also slower washout for fertility planning. Sexual side effects parallel finasteride. Used as the canonical dual 5AR inhibitor in mechanism studies.

3. Mechanism of Action

Dual 5-alpha-reductase type I and II inhibition; reduces plasma DHT by 90 to 95 percent. More complete DHT suppression than finasteride.

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 0.5 mg per oral administration daily. Rodent studies 0.1 to 1 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. Roehrborn CG, et al. The effects of dutasteride on prostate volume and DHT in men with BPH. Urology 2002.
2. Olsen EA, et al. The importance of dual 5-alpha-reductase inhibition in the treatment of male pattern hair loss. J Am Acad Dermatol 2006.
3. Bramson HN, et al. Unique preclinical characteristics of GG745, a dual 5-alpha-reductase inhibitor. J Pharmacol Exp Ther 1997.

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