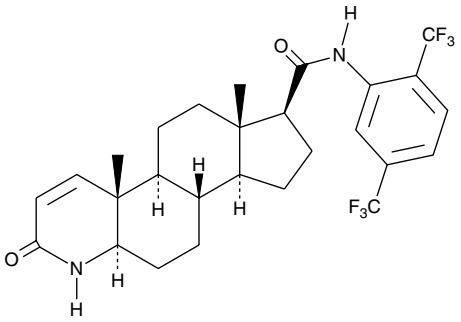
Product Information

Dutasteride

Item No. 15956

CAS Registry No.: 164656-23-9
Formal Name: N-[2,5-bis(trifluoromethyl)phenyl]-2,4aR,4bS,5,6,6aS,7S,8,9,9aS,9bS,10,11,11aR-tetradecahydro-4a,6a-dimethyl-2-oxo-1H-indeno[5,4-f]quinoline-7-carboxamide
Synonym: Avodart®
MF: $C_{27}H_{30}F_6N_2O_2$
FW: 528.5
Purity: ≥98%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid
UV/Vis.: λ_{max}: 206, 241, 278 nm



Laboratory Procedures

For long term storage, we suggest that dutasteride be stored as supplied at -20°C. It should be stable for at least two years.

Dutasteride is supplied as a crystalline solid. A stock solution may be made by dissolving the dutasteride in the solvent of choice. Dutasteride is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of dutasteride in ethanol and DMSO is approximately 10 mg/ml and approximately 30 mg/ml in DMF.

Dutasteride is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, dutasteride should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Dutasteride has a solubility of approximately 0.3 mg/ml in a 1:2 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

5α-reductase catalyzes the NADPH-dependent reduction of $\Delta^{4,5}$ double bonds in several steroid substrates, including testosterone (Item No. 15645), which is converted to dihydrotestosterone, the primary mediator of prostate growth.[1] The 5α-reductase enzymes responsible for the reduction of testosterone to dihydrotestosterone exist as two forms: type I, which occurs in the skin, liver, and ventral prostate and type II, which is expressed in ventral prostate, epididymis, and other reproductive tissues.[2] Dutasteride is a time-dependent, dual inhibitor of 5α-reductase (apparent K_is = 17 and 4.3 nM at 10 and 30 minute reaction times, respectively) that is frequently used in the treatment of benign prostatic hyperplasia.[3-4] Compared to finasteride (Item No. 14938), a selective inhibitor of type II 5α-reductase, dutasteride has a greater terminal half-life (1 *vs.* 14 hours in rat, respectively) and is more effective at decreasing serum levels of dihydrotestosterone at single doses > 10 mg.[4] Dutasteride has also been used as a strategy to reduce androgen levels related to prostate tumor burden on androgen receptor activity.[5]

References

1. George, F.W., Russell, D.W., and Wilson, J.D. *Proc. Natl. Acad. Sci. USA* **88(18)**, 8044-8047 (1991).
2. Flores, E., Bratoeff, E., Cabeza, M., *et al. Mini Rev. Med. Chem.* **3(3)**, 225-237 (2003).
3. Makridakis, N. and Reichardt, J.K. *J. Mol. Endocrinol.* **34(3)**, 617-623 (2005).
4. Bramson, H.N., Hermann, D., Batchelor, K.W., *et al. J. Pharmacol. Exp. Ther.* **282(3)**, 1496-1502 (1997).
5. Nacusi, L.P. and Tindall, D.J. *Nat. Rev. Urol.* **8(7)**, 378-384 (2011).

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