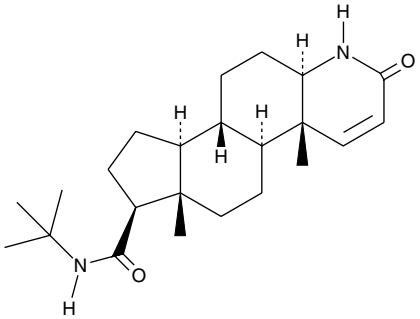
Product Information

Finasteride

Item No. 14938

CAS Registry No.: 98319-26-7
Formal Name: (4aR,4bS,6aS,7S,9aS,9bS,11aR)-N-(1,1-dimethylethyl)-2,4a,4b,5,6,6a,7,8,9,9a,9b,10,11,11a-tetradecahydro-4a,6a-dimethyl-2-oxo-1H-indeno[5,4-f]quinoline-7-carboxamide
Synonyms: MK 906, Propecia®, Proscar®
MF: $C_{23}H_{36}N_2O_2$
FW: 372.5
Purity: ≥95%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid



Laboratory Procedures

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

Finasteride is supplied as a crystalline solid. A stock solution may be made by dissolving the finasteride in the solvent of choice. Finasteride is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of finasteride in ethanol and DMF is approximately 25 mg/ml and approximately 16 mg/ml in DMSO.

Finasteride is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, finasteride should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Finasteride has a solubility of approximately 0.5 mg/ml in a 1:1 solution of ethanol: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, which is converted to dihydrotestosterone, the primary mediator of prostate growth.[1] The 5α-reductase enzymes responsible for the reduction of testosterone to dihydrotestosterone exists 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] Finasteride is a 4-azasteroid analog of testosterone that competitively blocks type II 5α-reductase activity (IC_{50} = 4.2 nM) with 100-fold greater affinity than for the type I enzyme.[2] It has been used in the treatment of benign prostatic hyperplasia, decreasing human prostatic dihydrotestosterone levels by 70-90% and reducing prostatic size.[2] However, at 10 µM finasteride has also been shown to induce the expression of Nrf2 and HO-1 proteins in androgen refractory prostrate PC-3 cells, which has been implicated in increased high-grade prostate tumor formation.[3] Also, at 0.1 µM finasteride can inhibit testosterone-induced type I procollagen and TGF-β1 expression in human scalp dermal fibroblasts in a model of androgenic alopecia.[4]

References

1. George, F.W., Russell, D.W., and Wilson, J.D. Feed-forward control of prostate growth: Dihydrotestosterone induces expression of its own biosynthetic enzyme, steroid 5α-reductase. *Proc. Natl. Acad. Sci. USA* **88(18)**, 8044-8047 (1991).
2. Flores, E., Bratoeff, E., Cabeza, M., *et al.* Steroid 5α-reductase inhibitors. *Mini Rev. Med. Chem.* **3(3)**, 225-237 (2003).
3. Yun, D.-K., Lee, J., and Keum, Y.-S. Finasteride increases the expression of hemoxygenase-1 (HO-1) and NF-E2-related factor-2 (Nrf2) proteins in PC-3 cells: Implication of finasteride-mediated high-grade prostate tumor occurence. *Biomol. Ther. (Seoul)* **21(1)**, 49-53 (2013).
4. Yoo, H.G., Kim, J.S., Lee, S.R., *et al.* Perifollicular fibrosis: Pathogenetic role in androgenetic alopecia. *Biol. Pharm. Bull.* **29(6)**, 1246-1250 (2006).

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Mailing address
1180 E. Ellsworth Road
Ann Arbor, MI
48108 USA

Phone
(800) 364-9897
(734) 971-3335

Fax
(734) 971-3640

E-Mail
custserv@caymanchem.com

Web
www.caymanchem.com