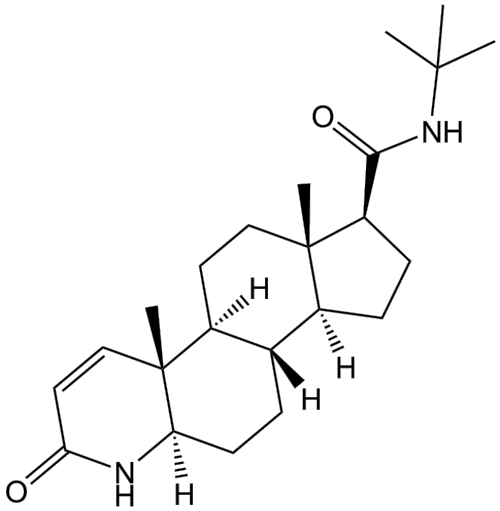
Product Data Sheet

Product Name: Finasteride
Cat. No.: GC16248

Chemical Properties

Cas. No.	98319-26-7		
Chemical Name	(1S,3aS,3bS,5aR,9aR,9bS,11aS)-N-tert-butyl-9a,11a-dimethyl-7-oxo-1,2,3,3a,3b,4,5,5a,6,9b,10,11-dodecahydroindeno[5,4-f]quinoline-1-carboxamide		
SMILES	CC12CCC3C(C1CCC2C(=O)NC(C)(C)C)CCC4C3(C=CC(=O)N4)C		
Formula	$C_{23}H_{36}N_2O_2$	M.Wt	372.54
Solubility	≥ 14.65 mg/mL in DMSO, ≥ 76.4 mg/mL in EtOH	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

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Cell lines	RWPE-1, LNCaP, PC3, DU145 (human prostate cancer cell line)
Preparation Method	Cells were maintained in RPMI 1640 medium supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO_2. Cells were treated with Finasteride at concentrations of 10μM and 50μM for 72 hours.
Reaction Conditions	10μM, 50μM; 72h
Applications	Finasteride significantly downregulated the activities of MMP2 and MMP9 in RWPE-1 and PC3 cells, and MMP2 in DU145 cells. Finasteride inhibited cell invasion in all tested cell lines.

Animal experiment [2]:

Animal models	Balb/c mice
Preparation Method	Chronic kidney disease (CKD) mouse models were generated by intraperitoneal injection of bovine serum albumin (BSA) at 30mg/30g body weight (with 15 and 25mg/30g BSA on the first 2 days). Mice were fed a high-fat diet (HFD) simultaneously with BSA injection for 4 weeks. In the last 2 weeks, mice were treated with Finasteride at 1.5mg/30g body weight via intragastric administration. Mice were sacrificed on Day 35 for evaluation of renal function and histological analysis.
Dosage form	1.5mg/30g body weight; i.g.
Applications	Finasteride significantly alleviated HFD-induced hyperlipidemia and renal injury in CKD mice, as evidenced by reduced levels of serum creatinine, blood urea nitrogen, and urinary protein.

Caution: Product has not been fully validated for medical applications. For research use only.
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References:
[1] Moroz A, Delella FK, Almeida R, et al. Felisbino SL. Finasteride inhibits human prostate cancer cell invasion through MMP2 and MMP9 downregulation. PLoS One. 2013 Dec 30;8(12):e84757.
[2] Wang Z, You L, Ren Y, et al. Finasteride Alleviates High Fat Associated Protein-Overload Nephropathy by Inhibiting Trimethylamine N-Oxide Synthesis and Regulating Gut Microbiota. Front Physiol. 2022 Aug 15;13:900961.

Background

Finasteride is a selective type II 5α-reductase inhibitor with an IC_{50} value of 4.2nM [1]. Finasteride lowers dihydrotestosterone (DHT) levels and is used to treat male pattern hair loss (androgenetic alopecia)[2]. Additionally, Finasteride can be used to treat female pattern hair loss in patients who have failed local minoxidil treatment [3]. Finasteride has been shown to significantly reduce prostate volume and improve urinary symptoms [4].

In vitro, treatment of human dermal papilla cells (DPCs) with Finasteride (10–100μM) for 5 days significantly increased cell aggregation behavior, upregulated the expression of stem cell transcription factors Nanog and Sox-2, and activated the protein kinase B (AKT), β-catenin, and integrin β-1 signaling pathways, thereby enhancing stem cell properties[5]. Treatment of human prostate cell lines RWPE-1, LNCaP, PC3, and DU145 with Finasteride (10μM and 50μM) for 72 hours significantly downregulated the activities of MMP2 and MMP9, while upregulating the expression of TIMP-2. Moreover, 50μM Finasteride significantly inhibited the invasive potential of all cell lines and also significantly inhibited the migration of RWPE-1 and LNCaP cells [6].

In vivo, $Ldlr^{-/-}$ mice treated orally with Finasteride (1000mg/kg) for 12 weeks showed significant reductions in total cholesterol and triglyceride levels, decreased atherosclerotic plaque area in the aortic arch and root, and reduced macrophage content and necrotic core area in the plaques [7]. In a protein-overload nephropathy chronic kidney disease (CKD) mouse model fed a high-fat diet (HFD), treatment with Finasteride (1.5mg/30g) via gavage for 2 weeks significantly lowered plasma trimethylamine N-oxide (TMAO) levels, improved renal injury, and regulated the composition and distribution of gut microbiota[8].

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[1] Flores E, Bratoeff E, Cabeza M, et al. Steroid 5alpha-reductase inhibitors. Mini Rev Med Chem. 2003 May;3(3):225-37.
[2] Piraccini BM, Blume-Peytavi U, Scarci F, et al. Topical Finasteride Study Group. Efficacy and safety of topical finasteride spray solution for male androgenetic alopecia: a phase III, randomized, controlled clinical trial. J Eur Acad Dermatol Venereol. 2022 Feb;36(2):286-294.
[3] Stout SM, Stumpf JL. Finasteride treatment of hair loss in women. Ann Pharmacother. 2010 Jun;44(6):1090-7.
[4] Tacklind J, Fink HA, Macdonald R, et al. Finasteride for benign prostatic hyperplasia. Cochrane Database Syst Rev. 2010 Oct 6;2010(10):CD006015.
[5] Rattanachitthawat N, Pinkhien T, Opanasopit P, et al. Finasteride Enhances Stem Cell Signals of Human Dermal Papilla Cells. In Vivo. 2019 Jul-Aug;33(4):1209-1220.
[6] Moroz A, Delella FK, Almeida R, et al. Felisbino SL. Finasteride inhibits human prostate cancer cell invasion through MMP2 and MMP9 downregulation. PLoS One. 2013 Dec 30;8(12):e84757.
[7] McQueen P, Molina D, Pinos I, et al. Finasteride delays atherosclerosis progression in mice and is associated with a reduction in plasma cholesterol in men. J Lipid Res. 2024 Mar;65(3):100507.
[8] Wang Z, You L, Ren Y, et al. Finasteride Alleviates High Fat Associated Protein-Overload Nephropathy by Inhibiting Trimethylamine N-Oxide Synthesis and Regulating Gut Microbiota. Front Physiol. 2022 Aug 15;13:900961.

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