Dutasteride

Cat. No.: HY-13613

CAS No.: 164656-23-9

Molecular Formula: $C_{27}H_{30}F_6N_2O_2$

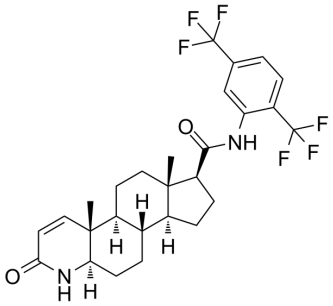
Molecular Weight: 528.53

Target: 5 alpha Reductase; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (63.06 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8920 mL	9.4602 mL	18.9204 mL
	5 mM	0.3784 mL	1.8920 mL	3.7841 mL
	10 mM	0.1892 mL	0.9460 mL	1.8920 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dutasteride (GG745) is a potent inhibitor of both 5α-reductase isozymes. Dutasteride may possess off-target effects on the androgen receptor (AR) due to its structural similarity to DHT[1].

IC_{50} & Target

IC50: 5 alpha-reductase[1]

In Vitro

Dutasteride inhibits ^{3}H-T conversion to ^{3}H-DHT and, as anticipated, inhibits T-induced secretion of PSA and proliferation. However the drug also inhibited DHT-induced PSA secretion and cell proliferation (IC_{50} approximately 1 μM)[1].
Dutasteride competes for binding the LNCaP cell AR with an IC_{50} approximately 1.5 μM. High concentrations of dutasteride (10-50 μM), but not finasteride, in steroid-free medium, resulted in enhanced cell death, possibly by apoptosis[1].
Dutasteride reduces cell viability and cell proliferation in both cell lines tested (androgen-responsive (LNCaP) and androgen-unresponsive (DU145) human prostate cancer (PCa)) [2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	GG745 has a terminal half-life of approximately 240 hr, and single doses of >10 mg decreased DHT levels significantly more than did single 5-mg doses of finasteride[3]. In placebo treated men without prostate cancer there was an 8.3% median increase in PSA at month 24 compared with -59.5% in those who received dutasteride, using doubled values to correct for dutasteride treatment[4]. Toxicity: Dutasteride may affect male fertility and steroid hormone dynamics. Therefore, a 21-day reproduction study was conducted to determine the effects of dutasteride (10, 32 and 100 μg/L) on fish reproduction. Exposure to dutasteride significantly reduced fecundity of fish and affected several aspects of reproductive endocrine functions in both males and females[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Pain. 2019 May;20(5):577-591.

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REFERENCES

[1]. Lazier CB, et al. Dutasteride, the dual 5alpha-reductase inhibitor, inhibits androgen action and promotes cell death in the LNCaP prostate cancer cell line. Prostate. 2004 Feb 1;58(2):130-44.

[2]. Biancolella M, et al. Effects of dutasteride on the expression of genes related to androgen metabolism and related pathway in human prostate cancer cell lines. Invest New Drugs. 2007 Oct;25(5):491-7.

[3]. Bramson HN, et al. Unique preclinical characteristics of GG745, a potent dual inhibitor of 5AR. J Pharmacol Exp Ther. 1997 Sep;282(3):1496-502.

[4]. Andriole GL, et al. Clinical usefulness of serum prostate specific antigen for the detection of prostate cancer is preserved in men receiving the dual 5alpha-reductase inhibitor dutasteride. J Urol. 2006 May;175(5):1657-62.

[5]. Margiotta-Casaluci L, et al. Mode of action of human pharmaceuticals in fish: the effects of the 5-alpha-reductase inhibitor, dutasteride, on reproduction as a case study. Aquat Toxicol. 2013 Mar 15;128-129:113-23.

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