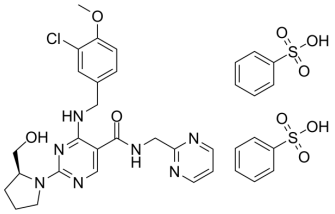
Avanafil dibenzenesulfonate

Cat. No.:	HY-18252A
CAS No.:	330784-48-0
Molecular Formula:	$C_{35}H_{38}ClN_7O_9S_2$
Molecular Weight:	800.3
Target:	Phosphodiesterase (PDE); NO Synthase; Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease; Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Avanafil (TA-1790) dibenzenesulfonate is a potent and selective phosphodiesterase-5 (PDE-5) inhibitor with IC_{50} values of 5.2 nM, 630 nM, 5700 nM, 6200 nM, 12000 nM, 27000 nM, 51000 nM and 53000 nM for PDE-5, PDE-6, PDE-4, PDE-10, PDE-8, PDE-7, PDE-2 and PDE-1, respectively. Avanafil dibenzenesulfonate activates NO/cGMP/PKG signaling-pathway to decrease loss in BMD, bone atrophy, and oxidative stress. Avanafil dibenzenesulfonate inhibits cyclic guanosine monophosphate (cGMP) hydrolysis and thus increases cGMP levels. Avanafil dibenzenesulfonate can be used for the research of erectile dysfunction and osteoporosis[1][2][3].

IC_{50} & Target

PDE5 5.2 nM (IC_{50})	PDE6 630 nM (IC_{50})	PDE4 5700 nM (IC_{50})	PDE10 6200 nM (IC_{50})
PDE7 27000 nM (IC_{50})	PDE2 51000 nM (IC_{50})	PDE1 53000 nM (IC_{50})	

In Vitro

Avanafil (TA-1790) dibenzenesulfonate (0.01-1000 μM) enhances by 45% for electrical field stimulation (1-20 Hz)-induced relaxation responses in corpus cavernosum strips from the diabetic group[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Avanafil (TA-1790) dibenzenesulfonate (10 mg/kg; p.o.; daily, for 30 d; male rat) increases angiogenesis in bone tissue via the activation of NO, cGMP and PKG (NO/cGMP/PKG) signaling-pathway and significantly decreases dexamethasone-induced loss in BMD, bone atrophy, and oxidative stress[1].

Avanafil (TA-1790) dibenzenesulfonate (10 μM; ICI; once, for 10 weeks) improves erectile responses in T2DM rats[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male rat model of glucocorticoid-induced osteoporosis (GIOP)[1]
Dosage:	10 mg/kg
Administration:	Oral administration; daily, for 30 days
Result:	Decreased the level of eNOS, NO, PDE-5, PICP, MDA, CoQ10/CoQ10H and 8-OHdG/10^8dG. Increased the level of cGMP, PKG, Cortisol and CTCP.

Animal Model:	Male rat model of glucocorticoid-induced osteoporosis (GIOP)[1]
Dosage:	10 mg/kg
Administration:	Oral administration; daily, for 30 days
Result:	Increased right femur trabecular bone thickness and epiphyseal bone width.

Animal Model:	Male T2DM Sprague Dawley rats[2]
Dosage:	10 μM
Administration:	Intracavernous injection; once, for 10 weeks
Result:	Increased in ICP/MAP in response to nerve stimulation and increased total ICP values.

CUSTOMER VALIDATION

- Chemrxiv. 2021, Jun 10.

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REFERENCES

[1]. Huyut Z, et, al. Effects of the Phosphodiesterase-5 (PDE-5) Inhibitors, Avanafil and Zaprinast, on Bone Remodeling and Oxidative Damage in a Rat Model of Glucocorticoid-Induced Osteoporosis. Med Sci Monit Basic Res. 2018 Mar 13;24:47-58.

[2]. Yilmaz D, et, al. The effect of intracavernosal avanafil, a newer phosphodiesterase-5 inhibitor, on neonatal type 2 diabetic rats with erectile dysfunction. Urology. 2014 Feb;83(2):508.e7-12.

[3]. Kotera J, et, al. Avanafil, a potent and highly selective phosphodiesterase-5 inhibitor for erectile dysfunction. J Urol. 2012 Aug;188(2):668-74.

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