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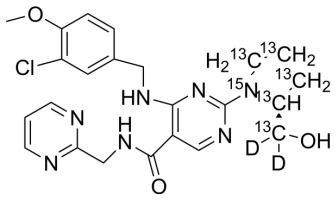
Avanafil-$^{13}C_5$,^{15}N,d_2

Cat. No.:	HY-18252S1
Molecular Formula:	$C_{18}{}^{13}C_5H_{24}D_2ClN_6{}^{15}NO_3$
Molecular Weight:	491.92
Target:	Endogenous Metabolite; NO Synthase; Phosphodiesterase (PDE); Isotope-Labeled Compounds
Pathway:	Metabolic Enzyme/Protease; Immunology/Inflammation; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Avanafil-$^{13}C_5$,^{15}N,d_2 is ^{15}N and deuterated labeled Avanafil (HY-18252). Avanafil (TA-1790) 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 activates NO/cGMP/PKG signaling-pathway to decrease loss in BMD, bone atrophy, and oxidative stress. Avanafil inhibits cyclic guanosine monophosphate (cGMP) hydrolysis and thus increases cGMP levels. Avanafil can be used for the research of erectile dysfunction and osteoporosis[1][2][3].
In Vitro	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1]. Avanafil (TA-1790) (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[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Avanafil (TA-1790) (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[2]. Avanafil (TA-1790) (10 μM; ICI; once, for 10 weeks) improves erectile responses in T2DM rats[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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.

[4]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

Caution: Product has not been fully validated for medical applications. For research use only.

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