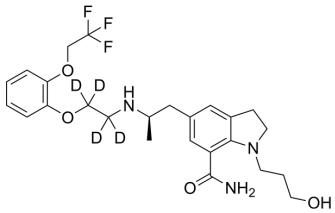
Silodosin-d_4

Cat. No.:	HY-10122S
CAS No.:	1426173-86-5
Molecular Formula:	$C_{25}H_{28}D_4F_3N_3O_4$
Molecular Weight:	499.56
Target:	Adrenergic Receptor; Isotope-Labeled Compounds
Pathway:	GPCR/G Protein; Neuronal Signaling; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Silodosin-d_4 is the deuterium labeled Silodosin. Silodosin (KAD 3213) is a potent, selective and orally active α1A-adrenergic receptor (α1A-AR) blocker. Silodosin exhibits high affinity for α1A-AR (Ki=0.036 nM), over 162-fold and 50-fold than for α1B-AR and α1D-AR with Ki values of 21 nM and 2.0 nM, respectively. Silodosin is an effective and well-tolerated agent, it can be used for the investigation of LUTS/BPH[1][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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

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

[2]. Maxime Rossi , Silodosin in the treatment of benign prostatic hyperplasia. Drug Des Devel Ther. 2010; 4: 291–297.

[3]. Villa L, et al. Effects by silodosin on the partially obstructed rat ureter in vivo and on human and rat isolated ureters.Br J Pharmacol. 2013 May;169(1):230-8.

[4]. Osman NI, et al.Silodosin : a new subtype selective alpha-1 antagonist for the treatment of lower urinary tract symptoms in patients with benign prostatic hyperplasia.Expert Opin Pharmacother. 2012 Oct;13(14):2085-96.

[5]. Kawahara T, et al. Silodosin inhibits the growth of bladder cancer cells and enhances the cytotoxic activity of cisplatin via ELK1 inactivation.Am J Cancer Res. 2015 Sep 15;5(10):2959-68. eCollection 2015.

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