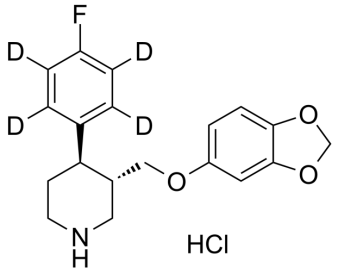
rel-Paroxetine-d_4 hydrochloride

Cat. No.:	HY-151216S
CAS No.:	1217683-35-6
Molecular Formula:	$C_{19}H_{17}D_4ClFNO_3$
Molecular Weight:	369.85
Target:	Serotonin Transporter; Autophagy
Pathway:	Neuronal Signaling; Autophagy
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	rel-Paroxetine-d_4 (hydrochloride) is an isotope-labeled Paroxetine hydrochloride (HY-B0492). Paroxetine hydrochloride is an orally active and selective serotonin-reuptake inhibitor, commonly prescribed as an GRK2 inhibitor with IC50 of 14 μM. Paroxetine hydrochloride can be used for the research of depressive disorder[1][2][3][4].
IC_{50} & Target	IC50: 14 μM (GRK2)[2]; Serotonin-reuptake[4]
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]. Paroxetine (1 μM and 10 μM; 4 h) distinctly restrains T cell migration induced by CX3CL1 through inhibiting GRK2[2]. Paroxetine (16 h) inhibits GRK2 induced activation of ERK in splenic T cells[2]. Paroxetine (10 μM) reduces pro-inflammatory cytokines in LPS-stimulated BV2 cells[3]. Paroxetine (0-5 μM) leads to a dose-dependent inhibition on LPS-induced production of TNF-α and IL-1β in BV2 cells[3]. Paroxetine also inhibits lipopolysaccharide (LPS)-induced nitric oxide (NO) production and inducible nitric oxide synthase (iNOS) expression in BV2 cells[3]. Paroxetine (5 μM) blocks LPS-induced JNK activation and attenuates baseline ERK1/2 activity in BV2 cells[3]. Paroxetine relieves microglia-mediated neurotoxicity, and suppresses LPS-stimulated pro-inflammatory cytokines and NO in primary microglial cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Paroxetine (15 mg/kg/d; p.o.; 15 d) obviously attenuates the symptoms of collagen-induced arthritis (CIA) rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Collagen-induced arthritis (CIA) model in rats (around 14-day-old)[2]
Dosage:	15 mg/kg
Administration:	Oral gavage; once daily; 15 days
Result:	Helped CIA rats to restore more body weight.

REFERENCES

[1]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019;53(2):211-223.

[2]. Wang Q, et al. Paroxetine alleviates T lymphocyte activation and infiltration to joints of collagen-induced arthritis. Sci Rep. 2017 Mar 28;7:45364.

[3]. Liu RP, et al. Paroxetine ameliorates lipopolysaccharide-induced microglia activation via differential regulation of MAPK signaling. J Neuroinflammation. 2014 Mar 12;11:47.

[4]. Hwang S, et al. Inhibitory effect of the selective serotonin reuptake inhibitor paroxetine on human Kv1.3 channels. Eur J Pharmacol. 2021 Dec 5;912:174567.

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