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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
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SKF-83566

Cat. No.: HY-103430A

CAS No.: 99295-33-7

Molecular Formula: $C_{17}H_{18}BrNO$

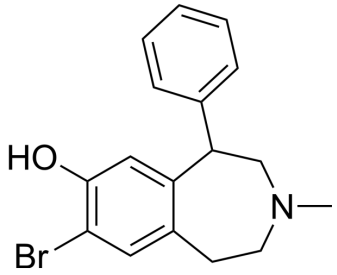
Molecular Weight: 332.23

Target: Dopamine Receptor; 5-HT Receptor; Adenylate Cyclase

Pathway: GPCR/G Protein; Neuronal Signaling

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 (100.32 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0100 mL	15.0498 mL	30.0996 mL
	5 mM	0.6020 mL	3.0100 mL	6.0199 mL
	10 mM	0.3010 mL	1.5050 mL	3.0100 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.52 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.52 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SKF-83566 is a potent, blood-brain permeable and orally active D1-like dopamine receptor (D1DR) antagonist and a weaker competitive antagonist at the vascular 5-HT_2 receptor (K_i=11 nM)[1][3]. SKF-83566 is a competitive DAT (dopamine transporter) inhibitor with an IC_{50} of 5.7 μM[2]. SKF-83566 also shows selective inhibition for adenylyl cyclase 2 (AC2) over AC_1 and AC_5 in the isolated rabbit thoracic aorta[4]. SKF-83566 can be used for research of parkinson's disease and nicotine craving alleviation[5].

IC_{50} & Target	D_1 Receptor	D_5 Receptor	5-HT_2 Receptor

			11 nM (Ki)

In Vitro

SKF-83566 (0.1 μM-10 μM) causes a concentration-dependent increase in peak evoked extracellular DA concentration ($[DA]_o$)?evoked by single-pulse stimulation, with a maximum 65% increase in peak evoked $[DA]_o$ with 5 μM. The EC_{50} value of this effect of SKF-83566 is 1.3 μM[2].

SKF-83566 inhibited [^{3}H]DA uptake with an IC_{50}?of 5.73 μM. Moreover, SKF-83566 more potently inhibits the binding of [^{3}H]CFT, with an IC_{50}?of 0.51 μM in [^{3}H]DA uptake and [^{3}H]CFT binding studies[2].

Similarly, in LLc-PK-rDAT cell, SKF-83566 also inhibits [^{3}H]CFT binding with an IC_{50}?of 0.77 μM in LLc-PK-rDAT cell membrane preparations[2].

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

In Vivo

SKF 83566 (oral administration; 20 μg/mL; 7 days) alone has no effects on altering LTP (115%). However, combinnation of SKF 83566 and nicotine significantly blocks the enhancement of long-term synaptic potentiation (LTP) induced by pretreatment with nicotine (SKF 83566+nicotine+cocaine, 120%; nicotine+cocaine, 143%)[1].

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

Animal Model:	Male C57BL6/J mice (6- to 9-wk-old)[1]
Dosage:	20 μg/mL (Together with nicotine for 7 d, followed by the injection of cocaine)
Administration:	Oral administration; 7 days
Result:	Blocked nicotine and cocaine-induced facilitation of LTP.

CUSTOMER VALIDATION

- Research Square Preprint. 2023 Oct 3.

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REFERENCES

[1]. Yan-You Huang, et al.D1/D5 Receptors and Histone Deacetylation Mediate the Gateway Effect of LTP in Hippocampal Dentate Gyrus.

[2]. Melissa A Stouffer, et al. SKF-83566, a D1-dopamine Receptor Antagonist, Inhibits the Dopamine Transporter. J Neurochem. 2011 Sep;118(5):714-20.

[3]. E H Ohlstein, et al. SCH 23390 and SK&F 83566 are antagonists at vascular dopamine and serotonin receptors. Eur J Pharmacol. 1985 Jan 22;108(2):205-8.

[4]. Jason M Conley, et al. Development of a high-throughput screening paradigm for the discovery of small-molecule modulators of adenylyl cyclase: identification of an adenylyl cyclase 2 inhibitor. J Pharmacol Exp Ther. 2013 Nov;347(2):276-87

[5]. Yan-You Huang, et al. D1/D5 receptors and histone deacetylation mediate the Gateway Effect of LTP in hippocampal dentate gyrus. Learn Mem. 2014 Feb 18;21(3):153-60. doi: 10.1101/lm.032292.113.

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