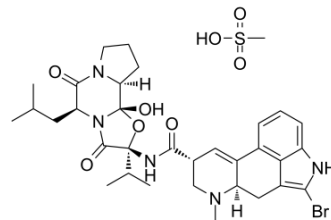


Bromocriptine mesylate

Cat. No.:	HY-12705A
CAS No.:	22260-51-1
Molecular Formula:	C ₃₃ H ₄₄ BrN ₅ O ₈ S
Molecular Weight:	750.7
Target:	Dopamine Receptor; Autophagy
Pathway:	GPCR/G Protein; Neuronal Signaling; Autophagy
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 75 mg/mL (99.91 mM; Need ultrasonic)
H₂O : 1.1 mg/mL (1.47 mM; ultrasonic and adjust pH to 3 with HCl)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.3321 mL	6.6605 mL	13.3209 mL
	5 mM	0.2664 mL	1.3321 mL	2.6642 mL
	10 mM	0.1332 mL	0.6660 mL	1.3321 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.08 mg/mL (2.77 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.08 mg/mL (2.77 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.08 mg/mL (2.77 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Bromocriptine mesylate is a potent dopamine D₂/D₃ receptor agonist, which binds D₂ dopamine receptor with pK_i of 8.05±0.2.

IC₅₀ & Target

pK_i: 8.05±0.2 (dopamine D₂ receptor)^[1]

In Vitro

Bromocriptine stimulates [³⁵S]-GTPγS binding at D₂ dopamine receptor expressed in CHO cells with pEC₅₀ of 8.15±0.05^[1]. Bromocriptine also is a strong inhibitor of brain nitric oxide synthase. The ergot alkaloid Bromocriptine (BKT) is found to act as a strong inhibitor of purified neuronal nitric oxide synthase (NOS) (IC₅₀=10±2 μM) whereas it is poorly active towards

inducible macrophage NOS ($IC_{50} > 100 \mu M$) [2]. Bromocriptine is found to inhibit the activity of at least one human cytochrome P450 enzyme. Bromocriptine is a potent inhibitor of CYP3A4 with a calculated IC_{50} value for the interaction of $1.69 \mu M$ [3].

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

In Vivo

Bromocriptine mesylate (2 mg/kg, i.p.) is administered for 7 days in groups of mice in forced swimming test (FST) and tail suspension test (TST). Bromocriptine group shows significant anti-immobility action as compared to control. When Bromocriptine administered 30 min after the last dose of 7 days MPE treatment and subjected to FST, this dopaminergic agonist produces significant and dose dependent potentiation of anti-immobility action of MPE (200 mg/kg, p.o.) as compared to MPE treatment alone. Bromocriptine treatment group shows a significant reduction of immobility time as compared to control. Bromocriptine administration after 7 days pretreatment with MPE (100 and 200 mg/kg, p.o.) shows significant and dose dependent potentiation of anti-immobility action of MPE as compared to MPE treatment alone [4]. Intracisternal administration of Bromocriptine decreases significantly the static mechanical allodynia (SMA) score compared to that of sham (saline-injected rats) and its effect lasted for 30 min. Intraperitoneal administration of Bromocriptine induces a significant, dose dependent (0.1 mg and 1 mg/kg) decrease in pain scores in CCI-IoN group when compared to sham and its effect lasted for 6 h. The highest dose induces the highest score decrease ($P < 0.01$). Bromocriptine effect lasts for 20 min. Intraperitoneal administration of Bromocriptine induces a significant dose dependent decrease in SMA score in CCI-IoN+6-OHDA lesioned group compared to that of sham. Its effect lasts for 6 h [5].

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PROTOCOL

Kinase Assay [1]

The [^{35}S]-GTP γ S binding assay is carried out. Cell membranes (25 \pm 75 μ g) are incubated in Buffer B containing 0.1 mM dithiothreitol (DTT) and 1 μ M GDP and drugs in a volume of 0.9 mL for 30 min at 30°C. This preincubation ensures that the agonists tested are at equilibrium when the [^{35}S]-GTP γ S (50 \pm 150 pM, final concentration) is added (in 100 μ L of Buffer B) to initiate the reaction. The assay mixture is incubated for a further 20 min unless otherwise stated. The assays are terminated by rapid filtration and bound radio-activity determined as described for the radio-ligand binding assays above. The total binding of [^{35}S]-GTP γ S is less than 20% of that added [1].

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Animal Administration [4][5]

Mice [4]

Swiss mice (20-25 g) of either sex (total 150) are used. Bromocriptine mesylate is used as dopamine receptor (D_2) agonist. Haloperidol is diluted in distilled water which is used for a vehicle of injection. Bromocriptine mesylate is dissolved in one drop of glacial acetic acid and made up to volume in distilled water. Imipramine is dissolved in 0.9% normal saline. Haloperidol (0.1 mg/kg, i.p.) and Bromocriptine mesylate (2 mg/kg, i.p.) are administered for 7 days in groups of mice in Forced Swimming Test (FST) and Tail Suspension Test (TST). Imipramine (10 mg/kg, p.o.) as a standard is administered in positive control groups for 7 days.

Rats [5]

Adult male Sprague-Dawley rats (N=112, 275-325 g) are used. Two weeks after the 6-OHDA injection, the animals are briefly (<3 min) anesthetized with 2% halothane using a mask and received for intracisternal administration Bromocriptine (7 μ g/kg dissolved in 5 μ L vehicle) or the vehicle alone (5 μ L of 0.9% saline). For i.p. injection we used Bromocriptine (1 mg/kg) and SKF81297 (3 mg/kg dissolved in 0.9% saline) concentrations. Following a recovery period (<2 min), the rats are placed in the observation field for 40 min period-test by a blind-experimenter.

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

CUSTOMER VALIDATION

- Nat Commun. 2020 Feb 18;11(1):941.
- Br J Pharmacol. 2021 Apr 26.

- J Ethnopharmacol. 2021 Mar 9;113994.
- Eur J Integr Med. 2021, 101322.

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REFERENCES

- [1]. Gardner B, et al. Agonist action at D2(long) dopamine receptors: ligand binding and functional assays. Br J Pharmacol. 1998 Jul;124(5):978-84.
 - [2]. Renodon A, et al. Bromocriptine is a strong inhibitor of brain nitric oxide synthase: possible consequences for the origin of its therapeutic effects. FEBS Lett. 1997 Apr 7;406(1-2):33-6.
 - [3]. Wynalda MA, et al. Assessment of potential interactions between dopamine receptor agonists and various human cytochrome P450 enzymes using a simple in vitro inhibition screen. Drug Metab Dispos. 1997 Oct;25(10):1211-4.
 - [4]. Rana DG, et al. Dopamine mediated antidepressant effect of Mucuna pruriens seeds in various experimental models of depression. Ayu. 2014 Jan;35(1):90-7.
 - [5]. Dieb W, et al. Nigrostriatal dopaminergic depletion increases static orofacial allodynia. J Headache Pain. 2016;17:11.
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Caution: Product has not been fully validated for medical applications. For research use only.

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