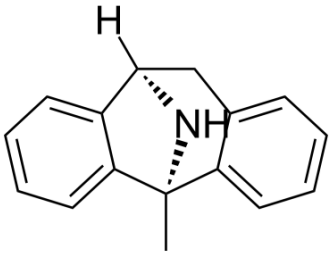
Dizocilpine

Cat. No.:	HY-15084B
CAS No.:	77086-21-6
Molecular Formula:	$C_{16}H_{15}N$
Molecular Weight:	221.3
Target:	iGluR
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Dizocilpine (MK-801), a potent anticonvulsant, is a selective and non-competitive NMDA receptor antagonist, with a K_d of 37.2 nM in rat brain membranes. Dizocilpine acts by binding to a site located within the NMDA associated ion channel and thus prevents Ca^{2+} flux[1][2].
IC_{50} & Target	Ki: 37.2 nM (NMDA receptor, in rat brain membrane)[1]
In Vitro	Dizocilpine (MK-801) progressively suppresses of current induced by NMDA. Mg^{2+} (10 mM) prevents Dizocilpine from blocking the N-Me-D-Asp-induced current, even when Dizocilpine is applied for a long time in the presence of NMDA. Dizocilpine blocks NMDA-activated single-channel activity in outside-out patches[3]. Dizocilpine (MK-801; <500 μM) inhibits activation of microglia induced by LPS with increased Cox-2 protein expression in BV-2 cells. Dizocilpine (<500 μM) reduces microglial TNF-α output with an EC_{50} of 400 μM in BV-2 cells[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Dizocilpine (MK 801) (1 mg/kg) treatment before each METH injection reduces the extent of DA depletion by 55% in striatal of mice. Dizocilpine (MK 801) (1 mg/kg) also attenuates the effects of METH on microglial activation in striatal of mice[4]. Dizocilpine ((+)-MK 801) (0.05, 0.2 mg/kg, i.p.) attenuates subsequent cocaine-primed reinstatement without disruption in rats. Dizocilpine (MK 801) (0.2 mg/kg, i.p.) prior to two reactivation sessions in the home cage shows no suppression on subsequent cocaine-primed reinstatement[5]. Dizocilpine (0.03, 0.1, 0.3 and 1 mg/kg, i.p.) significantly increases the ambulation of mice at 0.3 and 1 mg/kg, but not at 0.03 and 0.1 mg/kg[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Br J Pharmacol. 2020 Oct;177(20):4720-4733.
- Front Cell Dev Biol. 2020 Feb 4;8:24.
- Front Cell Neurosci. 2019 Jun 25;13:276.
- Front Neurosci. 2019 Nov 19;13:1225.
- Eur J Pharmacol. 2019 Aug 15;857:172427.

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REFERENCES

[1]. Wong EH, et al. The anticonvulsant MK-801 is a potent N-methyl-D-aspartate antagonist. Proc Natl Acad Sci U S A. 1986 Sep;83(18):7104-8.

[2]. Vardhan Reddy KH, et al. Convergent Strategy to Dizocilpine MK-801 and Derivatives. J Org Chem. 2018 Apr 6;83(7):4264-4269.

[3]. Huettner JE, et al. Block of N-methyl-D-aspartate-activated current by the anticonvulsant MK-801: selective binding to open channels. Proc Natl Acad Sci U S A. 1988 Feb;85(4):1307-11.

[4]. Thomas DM, et al. MK-801 and dextromethorphan block microglial activation and protect against methamphetamine-induced neurotoxicity. Brain Res. 2005 Jul 19;1050(1-2):190-8.

[5]. Brown TE, et al. The NMDA antagonist MK-801 disrupts reconsolidation of a cocaine-associated memory for conditioned place preference but not for self-administration in rats. Learn Mem. 2008 Dec 2;15(12):857-65.

[6]. Iijima Y, et al. Modification by MK-801 (dizocilpine), a noncompetitive NMDA receptor antagonist, of morphine sensitization: evaluation by ambulation in mice. Nihon Shinkei Seishin Yakurigaku Zasshi. 1996 Feb;16(1):11-8.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA