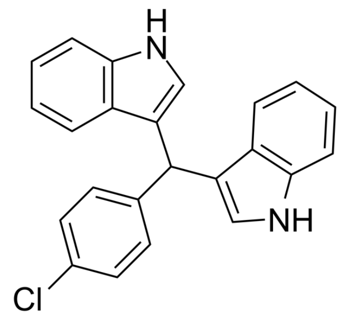
C-DIM12

Cat. No.:	HY-19808
CAS No.:	178946-89-9
Molecular Formula:	$C_{23}H_{17}ClN_2$
Molecular Weight:	356.85
Target:	Nuclear Hormone Receptor 4A/NR4A
Pathway:	Vitamin D Related/Nuclear Receptor
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (280.23 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8023 mL	14.0115 mL	28.0230 mL
	5 mM	0.5605 mL	2.8023 mL	5.6046 mL
	10 mM	0.2802 mL	1.4011 mL	2.8023 mL

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

BIOLOGICAL ACTIVITY

Description

C-DIM12 is a potent, orally active Nurr1 antagonist. C-DIM12 inhibits the tumor growth and autophagy, and induces the cell apoptosis. C-DIM12 has anti-inflammatory and neuroprotective effects, and can be used for cancer and neurological disease study[1][2][3].

IC_{50} & Target

Nurr1/NR4A2

In Vitro

C-DIM12 (15 μM, 3-5 day) increases cell proliferation and survival by inhibiting autophagy in MiaPaCa2 cells[1].

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

In Vivo

C-DIM12 (25 mg/kg for i.p., 14 day) modulates glial reactivity in MPTP-Induced Parkinsonism mice[2].

C-DIM12 (50-100 mg/kg for i.p., three times) attenuates brain inflammation and improves functional recovery after intracerebral hemorrhage in mice[3].

C-DIM12 (30 mg/kg for i.p., 30 day) inhibits tumor growth and autophagy, and induces apoptosis in NURR1-KO cells orthotopic xenograft[1].

Pharmacokinetic Analysis in C57BL/6 male mice[1]

Route	Organ	Dose (mg/kg)	Area under Curve (ng/mL*min)	$t_{1/2}$ (min)	C_{max} (ng/mL)
i.g.	Plasma	25	539,220	249	1120
i.g.	Brain	25	2,273,711	265	3622

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

Animal Model:	MPTP-induced C57BL/6 male Parkinsonism mice [2]
Dosage:	25 mg/kg/day, 14day
Administration:	Intragastric gavage (i.g.)
Result:	Protected against the loss of DA neurons in the substantia nigra pars compacta and DA terminals in the striatum, maintained a ramified phenotype in microglia, and suppressed activation of astrocytes.

Animal Model:	The ICR mice model of intracerebral hemorrhage induced by collagenase type VII[3]
Dosage:	50 and 100mg/kg/day at a 24-h interval, three times
Administration:	Orally administration
Result:	Improved the recovery of neurological function and prevented neuron loss in the hematoma, while suppressed activation of microglia/macrophages and expression of inflammatory mediators interleukin-6 and CC chemokine ligand 2. Preserved axonal structures in the internal capsule and axonal transport function. Decreased of iNOS mRNA expression.

Animal Model:	MiaPaCa2 cells (Ctrl and NURR1-KO) orthotopic xenograft tumor models[1]
Dosage:	30 mg/kg, 30 day
Administration:	Intraperitoneal injection (i.p.)
Result:	Inhibited tumor growth and ATG7 and ATG12 mRNA levels, and induced apoptosis.

CUSTOMER VALIDATION

- Research Square Preprint. 2021 Aug.

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REFERENCES

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[2]. Sean L. Hammond, et al. The Nurr1 Ligand,1,1-bis(3′-Indolyl)-1-(p-Chlorophenyl)Methane, Modulates Glial Reactivity and Is Neuroprotective in MPTP-Induced Parkinsonism. J Pharmacol Exp Ther. 2018 Jun; 365(3): 636–651.

[3]. Keita Kinoshita, et al. A Nurr1 ligand C-DIM12 attenuates brain inflammation and improves functional recovery after intracerebral hemorrhage in mice. Sci Rep. 2022; 12: 11009.

[4]. De Miranda BR, et al. The Nurr1 Activator 1,1-Bis(3'-Indolyl)-1-(p-Chlorophenyl)Methane Blocks Inflammatory Gene Expression in BV-2 Microglial Cells by Inhibiting Nuclear Factor κB. Mol Pharmacol. 2015 Jun;87(6):1021-34. doi: 10.1124/mol.114.095398. Epub 2015 Apr 9.

[5]. Hammond SL, et al. A novel synthetic activator of Nurr1 induces dopaminergic gene expression and protects against 6-hydroxydopamine neurotoxicity in vitro. Neurosci Lett. 2015 Oct 21;607:83-9.

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