Product Data Sheet

L-DOPA sodium

Cat. No.: HY-N0304A CAS No.: 63302-01-2 Molecular Formula:

Molecular Weight: 219.17

Target: Dopamine Receptor; Endogenous Metabolite

 $C_9H_{10}NNaO_4$

Pathway: GPCR/G Protein; Neuronal Signaling; Metabolic Enzyme/Protease

Please store the product under the recommended conditions in the Certificate of Storage:

Analysis.

BIOLOGICAL ACTIVITY

Description L-DOPA (Levodopa) sodium is an orally active metabolic precursor of neurotransmitters dopamine. L-DOPA sodium can cross the blood-brain barrier and is converted into dopamine in the brain. L-DOPA sodium has anti-allodynic effects, and can be used for Parkinson's disease research^{[1][2][3]}.

IC₅₀ & Target Human Endogenous Metabolite

In Vivo L-DOPA sodium (20 mg/kg; orally) reduces Rotenone-induced motor dysfunction^[3].

> L-DOPA sodium (0-100 mg/kg; orally) reverses tactile, cold and heat allodynia in neuropathic rat without any side effect in sprague-Dawley rats^[4].In adult common marmosets (Callithrix jacchus, 2-3 years old, 270-350 g), L-DOPA (20/5 mg/kg, p.o.) shows the T_{max} was 30 min in plasma and 60-90 min in extracellular fluid (ECF) of striatum. Mean C max was 20.3 μM in plasma and 442.9 nM in ECF of striatum, which is about 2.2% of that in plasma^[6].

Induction of dyskinesia model^[5]

Background

L-DOPA-induced dyskinesia results from a pulsatile stimulation of brain dopamine (DA) receptors, triggering a complex cascade of molecular and synaptic alterations within the basal ganglia^[5].

Specific Mmodeling Methods

Mice: C57Bl/6 mice?•?male?• 8 weeks (period: 21 days) Administration: 20 mg/kg?•?ip?•?once daily for 21 days

(1) sustained unilateral 6-OHDA injections in the striatum before starting treatment.

(2) Injection volume is 10mL/kg body weight.

Modeling Indicators

Behavioral changes: Shows developed abnormal involuntary movements (AIMs) affecting the head, trunk and forelimb on the side contralateral to the lesion.

- Correlated Product(s): Oxidopamine hydrochloride (HY-B1081)
- Opposite Product(s): Oxidopamine hydrobromide (HY-B1081A)

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

Animal Model:	C57BL/6J mice (7-week-old) ^[3]
Dosage:	20 mg/kg
Administration:	Orally
Result:	Reduced Rotenone-induced motor dysfunction.
Animal Model:	Sprague-Dawley rats ^[4]
Dosage:	10, 30, 50, 70, and 100 mg/kg
Administration:	Orally
Result:	Reverses tactile, cold and heat allodynia in neuropathic rat without any side effect.

CUSTOMER VALIDATION

- Int J Biol Macromol. 2020 Jun 15;153:88-99.
- Antioxidants (Basel). 2022, 11(7), 1317.
- Nutrients. 2022, 14(21), 4678
- Int J Mol Sci. 2022, 23(20), 12420.
- CNS Neurosci Ther. 2023 Apr 26.

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REFERENCES

[1]. Hyland K, et al. Aromatic L-amino acid decarboxylase deficiency: diagnostic methodology. Clin Chem. 1992 Dec;38(12):2405-10.

- [2]. Merims D, et al. Dopamine dysregulation syndrome, addiction and behavioral changes in Parkinson's disease. Parkinsonism Relat Disord. 2008;14(4):273-80. Epub 2007 Nov 7.
- [3]. Perez-Pardo P, et al. Additive Effects of Levodopa and a Neurorestorative Diet in a Mouse Model of Parkinson's Disease. Front Aging Neurosci. 2018 Aug 3;10:237.
- [4]. Park HJ, et al. Anti-allodynic effects of levodopa in neuropathic rats. Yonsei Med J. 2013 Mar 1;54(2):330-5.
- [5]. M Lundblad, et al. Pharmacological validation of a mouse model of I-DOPA-induced dyskinesia. Exp Neurol. 2005 Jul;194(1):66-75.
- [6]. Jie Zhang, et al. Pharmacokinetics of L-dopa in plasma and extracellular fluid of striatum in common marmosets. Brain Res. 2003 Dec 12;993(1-2):54-8.

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

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Page 3 of 3 www.MedChemExpress.com