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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
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- Gefahrgutzuschlag
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N-Acetyl-L-tryptophan

Cat. No.: HY-W011978

CAS No.: 1218-34-4

Molecular Formula: $C_{13}H_{14}N_2O_3$

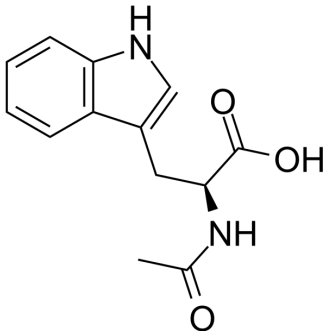
Molecular Weight: 246.26

Target: Endogenous Metabolite; Mitochondrial Metabolism; Neurokinin Receptor; Caspase; Interleukin Related

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

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (406.07 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.0607 mL	20.3037 mL	40.6075 mL
	5 mM	0.8121 mL	4.0607 mL	8.1215 mL
	10 mM	0.4061 mL	2.0304 mL	4.0607 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 (10.15 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.15 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.15 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

N-Acetyl-L-tryptophan is an antagonist of the neurokinin-1 receptor (NK-1R), disrupting the binding of substance P (SP) to NK-1R. This action provides neuroprotective effects, improving memory deficits and motor impairments. N-Acetyl-L-tryptophan is also an inhibitor of cytochrome c (Cytochrome c), and it exerts antioxidant and anti-inflammatory effects by inhibiting the expression of IL-1β and the activation of caspase-1. N-Acetyl-L-tryptophan holds promise for research in neurodegenerative and inflammatory diseases[1][2][3][4].

IC_{50} & Target	Microbial Metabolite	Human Endogenous Metabolite	NK1	Caspase-1
	IL-1β			

In Vitro

N-Acetyl-L-tryptophan (0.001–10 nM, 0.1–300 μM, 2 h) exhibits neuroprotective effects in a H_2O_2-induced amyotrophic lateral sclerosis model (cell lines: NSC-34 motoneurons and primary motor neurons.) induced by H_2O_2[1].
N-Acetyl-L-tryptophan (30 μM, 15 min-6 h) inhibits the secretion of Substance P (HY-P0201) and lL-1β and the activation of caspase-1 in NSC-34 motoneurons[1].
N-Acetyl-L-tryptophan (10 μM, 2 h) inhibits cell death in a H_2O_2-induced amyotrophic lateral sclerosis model by preventing the release of cytochrome c, Smac, and AIF from the mitochondria[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	H_2O_2-induced amyotrophic lateral sclerosis model (NSC-34 motoneurons and Primary motor neurons)
Concentration:	0.001, 0.01, 0.1, 1, 10 nM, 0.1, 1, 10, 30, 200, 300 μM
Incubation Time:	2 h
Result:	Significantly inhibited H_2O_2-induced cell death in NSC-34 motoneurons and primary motor neurons, with IC_{50} values of 0.3 μM and 16 nM, respectively.

Western Blot Analysis[1]

Cell Line:	H_2O_2-induced amyotrophic lateral sclerosis model (NSC-34 motoneurons)
Concentration:	10 μM
Incubation Time:	2 h
Result:	Effectively inhibited the release of cytochrome c/Smac/AIF from mitochondria into the cytoplasm.

In Vivo

N-Acetyl-L-tryptophan (10 mg/kg, i.p., single dose) confers hepatoprotection in an ischemia-reperfusion-induced Sprague-Dawley (SD) rat liver injury model by inhibiting excessive mitophagy[3].
N-Acetyl-L-tryptophan (0.5 mg/kg, s.c., once daily for 21 days) reduces the incidence of L-DOPA (HY-N0304)-induced dyskinesia (LID) in the hemi-parkinsonian rodent model (Sprague-Dawley rats)[3].
N-Acetyl-L-tryptophan (50 mg/kg, i.p., once daily for 28 days) can improve spatial memory deficits in the $AlCl_3$-induced Wistar rat dementia model[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Ischemia-reperfusion-induced liver injury model in Sprague-Dawley (SD) rats (200-220 g)[2]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection (i.p.), single dose
Result:	Reduced the expression of autophagy markers (Beclin1, LC3-II, ATG-7, and P62) in the ischemia-reperfusion-induced Sprague-Dawley (SD) rat liver injury model.

Animal Model:	L-DOPA (HY-N0304)-induced abnormal involuntary movements model in Sprague Dawley rats(230-270 g, before L-DOPA induction, a hemi-parkinsonian model was created by

	Oxidopamine hydrobromide (HY-B1081A))
Dosage:	0.5 mg/kg
Administration:	Subcutaneous injection (s.c.), once daily for 21 days
Result:	Significantly reduced the onset of dyskinesia.

REFERENCES

[1]. Sirianni A C, et al. N-acetyl-l-tryptophan, but not N-acetyl-d-tryptophan, rescues neuronal cell death in models of amyotrophic lateral sclerosis[J]. Journal of Neurochemistry, 2015, 134(5): 956-968.

[2]. Li H, et al. Inhibition of excessive mitophagy by N-acetyl-L-tryptophan confers hepatoprotection against Ischemia-Reperfusion injury in rats[J]. PeerJ, 2020, 8: e8665.

[3]. Thornton E, et al. The NK1 receptor antagonist N-acetyl-L-tryptophan reduces dyskinesia in a hemi-parkinsonian rodent model[J]. Parkinsonism & related disorders, 2014, 20(5): 508-513.

[4]. Fernandes J, et al. N-acetyl-L-tryptophan, a substance-P receptor antagonist attenuates aluminum-induced spatial memory deficit in rats[J]. Toxicology Mechanisms and Methods, 2018, 28(5): 328-334.

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