Emrusolmin

Cat. No.: HY-101855

CAS No.: 882697-00-9

Molecular Formula: $C_{16}H_{11}BrN_2O_2$

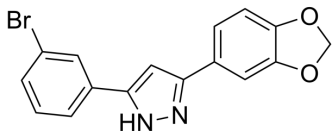
Molecular Weight: 343.17

Target: Amyloid-β

Pathway: Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (145.70 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9140 mL	14.5700 mL	29.1401 mL
5 mM	0.5828 mL	2.9140 mL	5.8280 mL
10 mM	0.2914 mL	1.4570 mL	2.9140 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 (7.29 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.29 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Emrusolmin (Anle138b), an oligomeric aggregation inhibitor, blocks the formation of pathological aggregates of prion protein (PrPSc) and of α-synuclein (α-syn). Emrusolmin strongly inhibits oligomer accumulation, neuronal degeneration, and disease progression in vivo. Emrusolmin has low toxicity and an excellent oral bioavailability and blood-brain-barrier penetration. Emrusolmin blocks Aβ channels and rescues disease phenotypes in a mouse model for amyloid pathology[1][2].

In Vitro

Oligomeric aggregates are presumed to be the key neurotoxic agent. Emrusolmin blocksthe formation of pathological aggregates of prion protein and of α-synuclein, which is deposited in Parkinson's disease and other synucleinopathies such as dementia with Lewy bodies and multiple system atrophy. Emrusolmin strongly inhibits all prion strains tested including BSE-derived and human prions. Emrusolmin shows structure-dependent binding to pathological aggregates and strongly

	inhibits formation of pathological oligomers both for prion protein and α-synuclein[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Emrusolmin shows structure-dependent binding to pathological aggregates and strongly inhibits formation of pathological oligomers in vitro and in vivo both for prion protein and α-synuclein[1]. Emrusolmin (0.6-2 g/kg; p.o.) modulates α‐synuclein oligomerization[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Two‐month‐old PLP‐hαSyn mice[3]
Dosage:	0.6 and 2 g/kg
Administration:	Oral
Result:	Prevented motor deficits and neurodegeneration in the PLP‐hαSyn mice.

REFERENCES

[1]. Wagner J, et al. Anle138b: a novel oligomer modulator for disease-modifying therapy of neurodegenerative diseases such as prion and Parkinson's disease. Acta Neuropathol. 2013 Jun;125(6):795-813.

[2]. Martinez Hernandez A, et al. The diphenylpyrazole compound anle138b blocks Aβ channels and rescues disease phenotypes in a mouse model for amyloid pathology. EMBO Mol Med. 2018;10(1):32-47.

[3]. Heras-Garvin A, et al. Anle138b modulates α-synuclein oligomerization and prevents motor decline and neurodegeneration in a mouse model of multiple system atrophy. Mov Disord. 2019;34(2):255-263.

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