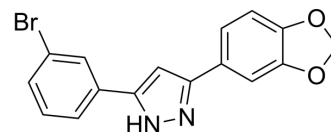


Emrusolmin

Cat. No.:	HY-101855
CAS No.:	882697-00-9
Molecular Formula:	C ₁₆ H ₁₁ BrN ₂ O 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.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- 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 (PrP^{Sc}) 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 A β 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

	<p>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.</p>	
In Vivo	<p>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.</p>	
	Animal Model:	Two-month-old PLP-haSyn mice ^[3]
	Dosage:	0.6 and 2 g/kg
	Administration:	Oral
	Result:	Prevented motor deficits and neurodegeneration in the PLP-haSyn 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 A β 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.

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

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