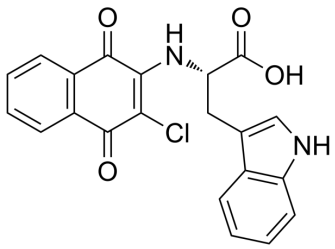
Cl-NQTrp

Cat. No.:	HY-138643
CAS No.:	185351-23-9
Molecular Formula:	$C_{21}H_{15}ClN_2O_4$
Molecular Weight:	394.81
Target:	Amyloid-β
Pathway:	Neuronal Signaling
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (633.22 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5329 mL	12.6643 mL	25.3286 mL
	5 mM	0.5066 mL	2.5329 mL	5.0657 mL
	10 mM	0.2533 mL	1.2664 mL	2.5329 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: ≥ 2.08 mg/mL (5.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cl-NQTrp signifcantly disrupts the preformed fbrillar aggregates of Tau-derived PHF6 (VQIVYK) peptide and full-length tau protein[1][2].

In Vitro

Cl-NQTrp efciently disassembled pre-formed PHF6 peptide fbrils[1].
Cl-NQTrp has the potential to induce conformational changes in PHF6 peptide oligomers[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Cl-NQTrp could be a unique potential therapeutic for AD since it targets aggregation of both Aβ and tau[2].
Cl-NQTrp significantly alleviates the shorter life span of htau-expressing flies, leading to 58% viability on day 29[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Virgin females, carrying either the eye GMR -Gal4 driver or the pan-neuronal driver elav^{c155} -Gal4 on chromosome X, were collected and crossed with males carrying UAS-h tau on the 2nd chromosome or with wild-type Oregon-R (OR) males as a control[2].
Dosage:	0.75 mg/mL.
Administration:	Dripped every other day.
Result:	Inhibited PHF6 aggregation and ameliorates eye neurodegeneration Drosophila overexpressing the human tau protein (htau).

REFERENCES

[1]. V Guru KrishnaKumar, et al. Mechanistic insights into remodeled Tau-derived PHF6 peptide fibrils by Naphthoquinone-Tryptophan hybrids. Sci Rep. 2018 Jan 8;8(1):71.

[2]. Moran Frenkel-Pinter, et al. Cl-NQTrp Alleviates Tauopathy Symptoms in a Model Organism through the Inhibition of Tau Aggregation-Engendered Toxicity. Neurodegener Dis. 2017;17(2-3):73-82.

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