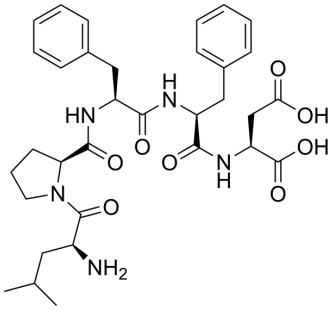
β-Sheet Breaker Peptide iAβ5



Cat. No.:	HY-P1047
CAS No.:	182912-74-9
Molecular Formula:	$C_{33}H_{43}N_5O_8$
Molecular Weight:	637.72
Sequence:	Leu-Pro-Phe-Phe-Asp
Sequence Shortening:	LPFFD
Target:	Amyloid-β
Pathway:	Neuronal Signaling
Storage:	Sealed storage, away from moisture and light, under nitrogen Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (78.40 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5681 mL	7.8404 mL	15.6809 mL
	5 mM	0.3136 mL	1.5681 mL	3.1362 mL
	10 mM	0.1568 mL	0.7840 mL	1.5681 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

β-Sheet Breaker Peptide iAβ5 is a potent degrader of cerebral amyloid-beta (Abeta). Abeta deposition is associatied with the Alzheimer disease (AD), due to its related toxicity linked to its beta-sheet conformation and/or aggregation. β-Sheet Breaker Peptide iAβ5 reproducibly induces in vivo disassembly of fibrillar amyloid deposits. Thus, β-Sheet Breaker Peptide iAβ5 prevents and/or reverses neuronal shrinkage caused by Abeta, and reduces the extent of interleukin-1beta positive microglia-like cells that surround the Abeta deposits. β-Sheet Breaker Peptide iAβ5 reduces the size and/or number of cerebral amyloid plaques in AD. β-Sheet Breaker Peptide iAβ5 labeled by hydrophobic benzyl alcohol (HBA) tag, can be used for quantitative assay by showing vivid blue color under acidic conditions[1][2][3].

In Vitro

β-Sheet Breaker Peptide iAβ5 (1.5 μg/μL; 7 days) incubates with Aβ1-42 (0.5 μg/μL) for 7 days, inhibits amyloid βprotein fibrillogenesis, disassembles preformed fibrils in vitro and prevents neuronal death induced by fibrils in cell culture[1]. β-Sheet Breaker Peptide iAβ5 (60 μM; 48 h) shows insignificant cytotoxicity in human neuroblastoma (IMR-32) cell treated

	with 50 μM aggregated Aβ1-42 for 2 days[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	β-Sheet Breaker Peptide iAβ5 (100 nmol/rat; Intra-amygdala injection; 7 days) coinjected with Aβ1-42 (5 nmol) into in the rat amygdala, blocks Aβ1-42 neurotoxicity in tissue culture, and amyloid fibril formation in the rat model[1]. β-Sheet Breaker Peptide iAβ5 (100 nmol/rat, 200 nmol/rat; Intra-amygdaloid injection; 7 days) followed by Aβ1-42 (5 nmol), induces disassembly of pre-existing Aβ fibrils in vivo, and leads to a reversal or prevention of Aβ-induced histopathological changes in rat model[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Sigurdsson EM, et al. In vivo reversal of amyloid-beta lesions in rat brain. J Neuropathol Exp Neurol. 2000 Jan;59(1):11-7.

[2]. Okada Y, et al. Acid-Triggered Colorimetric Hydrophobic Benzyl Alcohols for Soluble Tag-Assisted Liquid-Phase Synthesis. Org Lett. 2015 Sep 4;17(17):4264-7.

[3]. Soto C, et al. Beta-sheet breaker peptides inhibit fibrillogenesis in a rat brain model of amyloidosis: implications for Alzheimer's therapy. Nat Med. 1998 Jul;4(7):822-6.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA