Product Data Sheet

Product Name: β-Amyloid 22-35 (Amyloid β-Protein (22-35))
Cat. No.: GC30325

Chemical Properties

Cas. No.	144189-71-9		
SMILES	Glu-Asp-Val-Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met		
Formula	$C_{59}H_{102}N_{16}O_{21}S$	M.Wt	1403.62
Solubility	DMSO : 1.85 mg/mL (1.32 mM; ultrasonic and adjust pH to 5 with HCl); H_2O : 1.82 mg/mL (1.30 mM; ultrasonic and adjust pH to 3 with HCl)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Protocol

Cell experiment [1]:

Cell lines	Hippocampal neurons
Preparation Method	Hippocampal cells were cultured in serum-free Dulbecco's modified Eagle medium (DMEM) for 5 days prior to experimentation. Cells were treated with β-Amyloid 22-35 (10–40μg/mL) or β-Amyloid C-terminal amidated derivative (β22-35-NH_2) by adding 20μL of stock solution (2mg/mL in betaine buffer, pH 8.5) to 1.0mL culture medium.

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Reaction Conditions	10–40μg/mL; 3 days.
Applications	β-Amyloid 22-35 induced significant neurotoxicity in a dose-dependent manner, characterized by neurite fragmentation, shrinkage of cell bodies and nuclei, and increased lactate dehydrogenase (LDH) release.

Animal experiment [2]:

Animal models	Kunming mice
Preparation Method	Mice were intracerebroventricularly (ICV) injected with a single dose of aggregated β-Amyloid 22-35 (4nmol in 4μL saline) to induce dementia. Behavioral tests (water maze and shuttle box) were conducted 12 days post-injection, followed by 10-day treatment with P11-hEGF or hEGF (4μg/day) via intravenous (IV) or rectal administration.
Dosage form	4nmol/mouse; ICV; Single injection.
Applications	β-Amyloid 22-35 injection induced significant learning and memory deficits, characterized by prolonged latencies in water maze tests and increased avoidance time in shuttle box tests. Treatment with P11-hEGF (but not hEGF) rescued cognitive impairments, restored nestin expression, reduced GFAP overexpression, and promoted BrdU incorporation in hippocampal neurons, demonstrating recovery of neurogenesis and attenuation of β-Amyloid 22-35 induced pathology.

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References:
[1] Takadera T, Sakura N, Mohri T, et al. Toxic effect of a beta-amyloid peptide (beta 22-35) on the hippocampal neuron and its prevention. Neurosci Lett. 1993 Oct 14;161(1):41-4.
[2] Zhao BQ, Guo YR, Li XL, et al. Amelioration of dementia induced by Aβ 22-35 through rectal delivery of undecapeptide-hEGF to mouse brain. Int J Pharm. 2011 Feb 28;405(1-2):1-8.

Background

β-Amyloid 22-35 (Amyloid β-Protein (22-35)) is a 14-amino acid peptide fragment that can spontaneously aggregate to form β-sheet structures and exerts significant cytotoxicity on hippocampal neurons[1-2]. β-Amyloid 22-35 is applicable for studying the pathological mechanisms of Alzheimer's disease (AD), constructing drug screening models, and investigating related neurodegenerative diseases[3-4].

In vitro, β-Amyloid 22-35 (10–40μg/mL) was applied to cultured rat hippocampal neurons under serum-deprived conditions for 3 days. β-Amyloid 22-35 induced cytotoxicity, characterized by neurite fragmentation and increased lactate dehydrogenase release[5].

In vivo, β-Amyloid 22-35 (4nmol in 4μL; single injection) was administered to mice via intracerebroventricular injection. β-Amyloid 22-35 was shown to induce hippocampal neuronal damage and lead to learning and memory dysfunction, as evidenced by

prolonged latencies in water maze tests and impaired escape responses in shuttle box tests[6].

References:

[1] Chauhan V, Sheikh AM, Chauhan A, et al. Fibrillar amyloid beta-protein inhibits the activity of high molecular weight brain protease and trypsin. J Alzheimers Dis. 2005 Feb;7(1):37-44.
[2] Fawver JN, Duong KT, Wise-Scira O, et al. Probing and trapping a sensitive conformation: amyloid-β fibrils, oligomers, and dimers. J Alzheimers Dis. 2012;32(1):197-215.
[3] Yousefirad N, Kaygısız Z, Aydın Y. Amyloid beta peptide 22-35 induces a negative inotropic effect on isolated rat hearts. Int J Physiol Pathophysiol Pharmacol. 2016 Dec 25;8(4):146-151.
[4] Di Scala C, Yahi N, Lelièvre C, et al. Biochemical identification of a linear cholesterol-binding domain within Alzheimer's β amyloid peptide. ACS Chem Neurosci. 2013 Mar 20;4(3):509-17.
[5] Takadera T, Sakura N, Mohri T, et al. Toxic effect of a beta-amyloid peptide (beta 22-35) on the hippocampal neuron and its prevention. Neurosci Lett. 1993 Oct 14;161(1):41-4.
[6] Zhao BQ, Guo YR, Li XL, et al. Amelioration of dementia induced by Aβ 22-35 through rectal delivery of undecapeptide-hEGF to mouse brain. Int J Pharm. 2011 Feb 28;405(1-2):1-8.