

Amyloid beta-peptide(25-35)

Cat. No.:	HY-P0128		
CAS No.:	131602-53-4		
Molecular Formula:	C ₄₅ H ₈₁ N ₁₃ O ₁₄ S		
Molecular Weight:	1060.27		
Sequence:	Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met		
Sequence Shortening:	GSNKGAIIGLM		
Target:	Amyloid-β		
Pathway:	Neuronal Signaling		
Storage:	Powder	-80°C	2 years
		-20°C	1 year
	In solvent	-80°C	6 months
		-20°C	1 month

GSNKGAIIGLM

SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (47.16 mM; Need ultrasonic)			
		Solvent Concentration	Mass	
			1 mg	5 mg
	Preparing Stock Solutions	1 mM	0.9432 mL	4.7158 mL
		5 mM	0.9432 mL	1.8863 mL
		10 mM	0.0943 mL	0.4716 mL
	Please refer to the solubility information to select the appropriate solvent.			
In Vivo	1. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (2.36 mM); Clear solution			

BIOLOGICAL ACTIVITY

Description	Amyloid beta-peptide(25-35) is the fragment Aβ(25-35) of the Alzheimer's amyloid β-peptide, has shown neurotoxic activities in cultured cells.
In Vitro	The amino acid sequence of Aβ(25-35) peptide is NH ₂ -Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met-COOH, where the first Gly represents the amino acid 25 and the last Met represents the amino acid 35. Amyloid beta-peptide(25-35) is also investigated in gel state for the first time. Comparative studies are also carried out using vibrational absorption and ECD. The conformational preference of Aβ(25-35) peptide film is also investigated using vibrational absorption and VCD spectroscopy ^[1] . Amyloid beta-peptide(25-35) induces apoptotic effects on isolated brain mitochondria and

	the redox state of methionine-35, plays a key role in the induction of programmed cellular death pathways and toxic events ^[2] .
In Vivo	Rats are injected with A β _{25–35} peptide intracerebroventricularly and compound Danshen (CDS) are subsequently administered once daily for 23 days. Rats' behavior is monitored using Morris water maze and passive avoidance. Real time PCR and Western blotting are used in determining amyloid precursor protein (APP), β -site APP cleaved enzyme-1 (BACE1), Presenilin-1 (PS1), Insulin-degrading enzyme (IDE) and neprilysin (NEP) in hippocampus. The Alzheimer' s disease (AD) model group present with spatial learning and memory impairments. CDS and donepezil administration significantly ameliorate the A β _{25–35} peptide-induced memory impairment in both Morris water maze (P < 0.05) and passive avoidance task (P < 0.01) compared to the AD model group ^[3] .

PROTOCOL

Cell Assay ^[2]

Cell viability is determined by a modified MTS assay, which is based on the conversion of Tetrazolium salt by mitochondrial dehydrogenase to a formazan product spectrophotometrically measurable at 490 nm. PC12 cells are plated in 96-well plates at a density of 10 000 cells/well and maintained for 16 h in complete medium. Cells are then incubated in the absence (control) and presence of 40 μ M A β (31-35) and A β (25-35) with reduced, oxidized and norleucine-substituted methionine-35 staurosporine 10 μ M is used as positive control of 100% of cellular death. After 48 h of peptide-incubation, 20 μ L of MTS reagent (2.0 mg/mL) is added to each well. The cells are then incubated for 30-45 min at 37 °C in a 5% CO₂ incubator. The reaction is stopped by adding 25 μ L of 10% SDS. The plates are read with a microplate reader at 490 nm. Each data point is obtained using a triplet-well assay^[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration ^[3]

Rats^[3]

Fifty-four Male Sprague-Dawley rats (2 months old, 300-350 g) are used. Amyloid beta-peptide(25-35) is dissolved in sterile distilled water at a concentration of 1 mg/mL as a stocking solution. Animals are infused with 5 μ L/side of sterile distilled water (control), aggregated A β ₂₅₋₃₅ (2 μ g/ μ L), into bilateral cerebral lateral ventricles at a rate of 1 μ L/min; the needle is left in place for 5 min. Then the needles are removed and rats are kept on a warm pad until they are awakened. To determine the neuroprotective effect on AD rats, the A β ₂₅₋₃₅ treated rats are treated with CDS of different doses and Donepezil once daily for 23 days (including duration of behavior test). Experiment is performed to test the effect of CDS on A β ₂₅₋₃₅-induced memory impairment using Morris water-maze and step-through passive avoidance tasks. Specifically, all of the rats are randomly divided into 6 groups for the experiment: (a) Vehicle 1 (for A β ₂₅₋₃₅)+vehicle 2 (for CDS and Donepezil), (b) A β ₂₅₋₃₅+vehicle 2, (c) A β ₂₅₋₃₅+CDS (130 mg/kg), (d) A β ₂₅₋₃₅+CDS (260 mg/kg), (e) A β ₂₅₋₃₅+CDS (520 mg/kg), (f) A β ₂₅₋₃₅+Donepezil (0.5 mg/kg). One day after cerebroventricular microinfusions of A β ₂₅₋₃₅ (10 μ g/side) or its vehicle, rats are treated (i.g.) with CDS or Donepezil or vehicle 2, once daily for 14 days prior to the beginning of Morris water maze, followed by passive avoidance task.

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CUSTOMER VALIDATION

- Biomed Res Int. 2019 Apr 16;2019:4690280.

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REFERENCES

[1]. D'Ursi AM, et al. Solution structure of amyloid beta-peptide (25-35) in different media. J Med Chem. 2004 Aug 12;47(17):4231-8.

[2]. Clementi ME, et al. Abeta(31-35) and Abeta(25-35) fragments of amyloid beta-protein induce cellular death throughapoptotic signals: Role of the redox state of methionine-35. FEBS Lett. 2005 May 23;579(13):2913-8.

[3]. Liu M, et al. Cognitive improvement of compound danshen in an A β 25-35 peptide-induced rat model of Alzheimer's disease. BMC Complement Altern Med. 2015 Oct 23;15:382.

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