**Amyloid beta-peptide(25-35)**

**Cat. No.:** HY-P0128  
**CAS No.:** 131602-53-4  
**Molecular Formula:** C₄₅H₈₁N₁₃O₁₄S  
**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

### SOLVENT & SOLUBILITY

**In Vitro**  
**DMSO :** 50 mg/mL (47.16 mM; Need ultrasonic)  

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Mass (mL)</th>
<th>Solvent Concentration</th>
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<th>Solvent Concentration</th>
<th>Mass (mL)</th>
<th>Solvent Concentration</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>1 mM</td>
<td>1 mg</td>
<td>5 mg</td>
<td>10 mg</td>
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<td></td>
<td></td>
<td>0.9432 mL</td>
<td>4.7158 mL</td>
<td>9.4316 mL</td>
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<tr>
<td></td>
<td></td>
<td>0.1886 mL</td>
<td>0.9432 mL</td>
<td>1.8863 mL</td>
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<tr>
<td></td>
<td></td>
<td>0.0943 mL</td>
<td>0.4716 mL</td>
<td></td>
<td>0.9432 mL</td>
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</tbody>
</table>

*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: ≥ 2.5 mg/mL (2.36 mM); Clear solution
2. 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 NH₂-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 Aβ25–35 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 nepriysin (NEP) in hippocampus. The Alzheimer’s disease (AD) model group present with spatial learning and memory impairments. CDS and donepezil administration significantly ameliorate the Aβ25–35 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 μM Aβ(31-35) and Aβ(25-35) with reduced, oxidized and norleucine-substituted methionine-35 staurosporine 10 μM is used as positive control of 100% of cellular death. After 48 h of peptide-incubation, 20 μL 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 μL 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 μL/side of sterile distilled water (control), aggregated Aβ25–35 (2 μg/μL), into bilateral cerebral lateral ventricles at a rate of 1 μL/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 Aβ25–35 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 Aβ25–35-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 Aβ25–35)+vehicle 2 (for CDS and Donepezil), (b) Aβ25–35+vehicle 2, (c) Aβ25–35+CDS (130 mg/kg), (d) Aβ25–35+CDS (260 mg/kg), (e) Aβ25–35+CDS (520 mg/kg), (f) Aβ25–35+Donepezil (0.5 mg/kg). One day after cerebroventricular microinfusions of Aβ25–35 (10 μg/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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