β-Amyloid (31-35)

Cat. No.: HY-P1517

CAS No.: 149385-65-9

Molecular Formula: $C_{25}H_{47}N_5O_6S$

Molecular Weight: 545.74

Sequence: Ile-Ile-Gly-Leu-Met

Sequence Shortening: IIGLM

Target: Amyloid-β

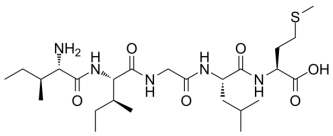
Pathway: Neuronal Signaling

Storage: Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (9.16 mM; ultrasonic and adjust pH to 7 with HCl)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8324 mL	9.1619 mL	18.3237 mL
	5 mM	0.3665 mL	1.8324 mL	3.6647 mL
	10 mM	---	---	---

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.81 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.08 mg/mL (3.81 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

β-Amyloid (31-35) is the shortest sequence of native Amyloid-β peptide that retains neurotoxic activity.

In Vitro

β-Amyloid (31-35) is a functional cytotoxic domain of Aβ peptide. β-Amyloid (31-35) increases the phosphorylation of biotinylated Aβ(1-40), enhances CDK-1 activity, and also inhibits binding of Aβ to cyclin B1. β-Amyloid (31-35) is cytotoxic, and such an effect can be inhibited by olomoucine in differentiated human teratocarcinoma cell line, Ntera 2/cl-D1 (NT-2) neurons[1].

β-Amyloid Aggregation Guidelines (Following is our recommended protocol. This protocol only provides a guideline, and

should be modified according to your specific needs).
1. Solid Aβ peptide was dissolved in cold hexafluoro-2-propanol (HFIP). The peptide was incubated at room temperature for at least 1h to establish monomerization and randomization of structure.
2. The HFIP was removed by evaporation, and the resulting peptide was stored as a film at -20 or -80 °C.
3. The resulting film was dissolved in anhydrous DMSO at 5 mM and then diluted into the appropriate concentration and buffer (serum- and phenol red-free culture medium) with vortexing.
4. Next, the solution was age 48h at 4-8 °C. The sample was then centrifuged at 14000g for 10 min at 4-8 °C; the soluble oligomers were in the supernatant. The supernatant was diluted 10-200-fold for experiments.
Methods vary depends on the downstream applications.
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Milton NG, et al. The amyloid-beta peptide binds to cyclin B1 and increases human cyclin-dependent kinase-1 activity. Neurosci Lett. 2002 Apr 5;322(2):131-3.

[2]. Misiti F, et al. Fragment 31-35 of beta-amyloid peptide induces neurodegeneration in rat cerebellar granule cells via bax gene expression and caspase-3 activation. A crucial role for the redox state of methionine-35 residue. Neurochem Int. 2006 Oct;49(5):

[3]. M Jesús Pérez de Vega, et al. Synthesis and biological properties of beta-turned Abeta(31-35) constrained analogues. Bioorg Med Chem Lett. 2008 Mar 15;18(6):2078-82.

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