β-Amyloid (1-40)

Cat. No.: HY-P0265

CAS No.: 131438-79-4

Molecular Formula: $C_{194}H_{295}N_{53}O_{58}S$

Molecular Weight: 4329.82

DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

Sequence: Asp-Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr-Glu-Val-His-His-Gln-Lys-Leu-Val-Phe-Phe-Ala-Glu-Asp-Val-Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met-Val-Gly-Gly-Val-Val

Sequence Shortening: DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

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

H_2O : 100 mg/mL (23.10 mM; Need ultrasonic)

DMSO : 100 mg/mL (23.10 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.2310 mL	1.1548 mL	2.3096 mL
	5 mM	0.0462 mL	0.2310 mL	0.4619 mL
	10 mM	0.0231 mL	0.1155 mL	0.2310 mL

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

BIOLOGICAL ACTIVITY

Description

β-Amyloid (1-40) is a primary protein in plaques found in the brains of patients with Alzheimer's disease.

In Vitro

β-Amyloid (1-40) and (1-42) are major components of senile plaque amyloids, are physiological peptides present in the brain, cerebrospinal fluid (CSF) and plasma. The levels of CSF β-Amyloid (1-40) and (1-42) show a U-shaped natural course in normal aging[1].Chronic infusion of beta-amyloid (1-40) for 14 days into the rat cerebroventricle decreased the activity of soluble protein kinase C (PKC) in the hippocampus. Subcellular translocation of PKC to membrane fraction in hippocampal slices of rats treated with beta-amyloid (1-40) is completely abolished under acute stimulation with 0.5 microM phorbol-dibutyrate (PDBu)[2].

The further aggregation of β-Amyloid (1-40)

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.
In Vivo	Chronic infusion of β-Amyloid (1-40) into rat cerebroventricle leads to deficit in spatial and non-spatial memory formation[2]. Chronic treatment of β-Amyloid (1-40) does not change lever-pressing performance significantly, but performance declined significantly 30 days after termination of the chronic daily regimen. The soluble unaggregated form of β-Amyloid (1-40), injected into the dorsal hippocampus, does not appear to have behavioral effects on performance or short-term working memory in rats, but multiple repeat injections produced performance decrements several weeks later. Repeated injection of β-Amyloid (1-40) through indwelling cannulae shows promise for development of an animal model of Alzheimer's disease[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3]	Rats: HPLC buffer insures the β-Amyloid (1-40) does not aggregate in solution prior to injection, β-Amyloid (1-40) and vehicle are bilaterally infused into the hippocampus, 20 min before experimental sessions, in volumes of 1, 2 and 3 μL per side, at a rate of <1 μL/min. Different volumes of the 1 μM solution are used. Volumes (doses) are given in random order and at least three sham-injection sessions are interposed between β-Amyloid (1-40) or vehicle injections. All rats receive all doses of β-Amyloid (1-40) under the acute injection regimen[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2023 Nov 10:e2303402.
- Nano Res. 28 April 2022.
- Appl Surf Sci. 2023 Sep 9, 158427.
- Nanomaterials. 2022 Nov 16;12(22):4031.
- Evid Based Complement Alternat Med. 2022 Sep 9;2022:3100621.

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REFERENCES

[1]. Shoji M, et al. Cerebrospinal fluid Abeta40 and Abeta42: natural course and clinical usefulness. Front Biosci. 2002 Apr 1;7:d997-1006.

[2]. Cleary J, et al. Beta-amyloid(1-40) effects on behavior and memory. Brain Res. 1995 Jun 5;682(1-2):69-74.

[3]. Olariu A, et al. Memory impairment induced by chronic intracerebroventricular infusion of beta-amyloid (1-40) involves downregulation of protein kinase C. Brain Res. 2002 Dec 13;957(2):278-86.

Caution: Product has not been fully validated for medical applications. For research use only.

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