β-Amyloid (1-40) (TFA)

Cat. No.: HY-P0265A

Molecular Formula: $C_{194}H_{295}N_{53}O_{58}S.C_2HF_3O_2$

Molecular Weight: 4443.84

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 DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV (TFA salt)

Sequence Shortening: DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

Target: Amyloid-β

Pathway: Neuronal Signaling

Storage: Sealed storage, away from moisture and light

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 (22.50 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.2250 mL	1.1252 mL	2.2503 mL
	5 mM	0.0450 mL	0.2250 mL	0.4501 mL
	10 mM	0.0225 mL	0.1125 mL	0.2250 mL

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

BIOLOGICAL ACTIVITY

Description

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

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].

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

After intraperitoneal injection of Ferric nitrilotriacetate into mice, it accumulates significantly in the liver and pancreas. Ferric nitrilotriacetate can be used in animal modeling to develop kidney tumor and diabetes models[1][2][3][4].

Induction of Alzheimer's disease[2]

Background

β-Amyloid (1-40) induces Alzheimer's disease by accumulating in the brain and being neurotoxic.

Specific Mmodeling Methods

Rat: Wistar • male • 280-320 g
Administration: 0, 3, 30, 300 pmol • connection of cannulae to modified micro-osmotic pumps for infusion • 2 weeks

Note

(1) Dissolve β-Amyloid (1-40) in 35% acetonitrile/0.1% trifluoroacetic acid (TFA) .
(2) In each group of 7 rats, a catheter was placed into the left ventricle on day 1. A water maze task was performed on days 9-13 after the start of infusion. At the end of the behavioural experiments, 4 rats from each group were taken from each group and processed by severing their heads for ChAT activity assay. Three rats were taken for histochemical studies.
(3) In histochemical studies, rats were anaesthetised and executed by transaortic perfusion fixation, first in cold saline and then in 4% paraformaldehyde and 0.1 M sodium phosphate buffer. Brains were removed and fixed in the same fixative for 12 hours. Frozen brain tissue was cut at 20 μm using a cryostat and the periventricular region was collected.

Modeling Indicators

Molecular changes: The activity of acetyltransferase (ChAT) in the frontal cortex and hippocampus is reduced, and cholinergic neuron dysfunction occurs.
Organizational changes: Accumulation of β-Amyloid in the hippocampus and cerebral cortex.
Phenotypic observation: Memory impairment occurs.

Correlated Product(s): /

Opposite Product(s): /

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.
- Brain Res. 2024 Apr 10:1835:148932.

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REFERENCES

[1]. Nitta A, et al. β-Amyloid protein-induced Alzheimer's disease animal model[J]. Neuroscience letters, 1994, 170(1): 63-66.

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

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