Product Data Sheet

Product Name: β-Amyloid (1-42), human TFA
Cat. No.: GC16243

Chemical Properties

Cas. No.	107761-42-2		
Formula	$C_{203}H_{311}N_{55}O_{60}S$	M.Wt	4514.08
Solubility		Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	β-Amyloid (1-42), human TFA		

Protocol

Cell experiment [1]:

Cell lines	PC12, cerebral cortex neurons

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Preparation Method	β-Amyloid (1-42), human TFA was dissolved in dimethyl sulfoxide (DMSO) at a concentration of 1 mM and pre-incubated at 37°C for 7 days to promote aggregation and then diluted in medium, then oligomerized β-Amyloid (1-42) (equivalent to 1 mM peptides) were prepared for β-Amyloid (1-42) insult experiments. For PC12 cellular AD model construction, PC12 cells were firstly cultured in 20 ng/ml nerve growth factor (NGF) and 10% FBS for 72 h at 37°C with 95% air and 5% CO2 to promote PC12 cells differentiation, and then 1 uM of oligomerized β-Amyloid (1-42) were added for 24 h to build PC12 cellular AD models. For cellular AD model of cerebral cortex neurons, 1 mM of oligomerized β-Amyloid (1-42) peptides was added in primary cerebral cortex neurons for 24 h to build cellular AD model of cerebral cortex neurons.
Reaction Conditions	1 uM for 24 h
Applications	Cell viability was reduced in β-Amyloid (1-42) insult group compared with a control group in NGF stimulated PC 12 cells and primary cerebral cortex neurons from rat embryo cells, indicating successes in the construction of cellular AD models.

Animal experiment [2]:

Animal models	Male Wistar rats weighing 210-230 g

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Preparation Method	AD model was induced by β-Amyloid (1-42) dissolved in normal saline at the concentration of 4 μg/μl. The solution was kept at room temperature for 3 days before administration75. β-Amyloid (1-42) or saline was injected in a volume of 2 μl over 5 min via a microsyringe pump connected to the 25-gauge stainless steel needle bilaterally into the lateral cerebral ventricles according to stereotaxic coordination.
Dosage form	4 μg/μl, 2 μl, lateral cerebral ventricle injection
Applications	Immunostaining of β-Amyloid (1-42) of entorhinal cortex (EC), and dorsal hippocampus (dHPC) sections demonstrated a high level of Aβ plaques in β-Amyloid (1-42) animals compared to the saline group.

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References:

[1]: Yang H, Wang H, Shang H, et al. Circular RNA circ_0000950 promotes neuron apoptosis, suppresses neurite outgrowth and elevates inflammatory cytokines levels via directly sponging miR-103 in Alzheimer's disease[J]. Cell Cycle, 2019, 18(18): 2197-2214.
[2]:Salimi M, Tabasi F, Abdolsamadi M, et al. Disrupted connectivity in the olfactory bulb-entorhinal cortex-dorsal hippocampus circuit is associated with recognition memory deficit in Alzheimer's disease model[J]. Scientific Reports, 2022, 12(1): 1-13.

Background

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Background

β-Amyloid (1-42), human TFA is a 42-amino acid peptide. Alzheimer's disease (AD) is characterized phenotypically by memory impairment, neurochemically by accumulation of β-amyloid peptide (such as β-Amyloid (1-42)) and morphologically by an initial loss of nerve terminals in cortical and hippocampal regions [1]. the abnormal production of soluble forms of β-amyloid peptides (Aβ), such as β-Amyloid (1-42), have been proposed as a major culprit in AD [2].

β-Amyloid (1-42) can impair synaptic function, typified by its ability to affect synaptic plasticity [3], and trigger events leading to a loss of viability of synapses[4], and leads to memory impairment [5]. The intracerebroventricular administration of β-Amyloid (1-42) has been proposed as a model of AD [6].

β-Amyloid (1-42) (100 μg/ml) for 24 h cell viability ofSH-SY5Y cells dropped to about 50%, β-Amyloid (1-42)-induced cell apoptosis could be completely prevented by EGb761 at 100 μg/ml and to a lesser extent, by quercetin (1.5 μg/ml) and ginkgolide B (10 μg/ml) [7].

β-Amyloid (1-42) (icv. 2 nmol in 4 μl) caused a predominant loss of glutamatergic and cholinergic markers [1].β-Amyloid (1-42) was combined with inducers of oxidative stress to induce neuronal cell death, amyloid deposits, gliosis and memory impairment following a 4 week intracerebroventricular infusion. Oxidative stress was induced using the pro-oxidative cation Fe2+ and the glutathione synthesis inhibitor buthionine sulfoximine (BSO) [8].

References:
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[2].Hardy J, Selkoe D J. The amyloid hypothesis of Alzheimer's disease: progress and problems on the road to therapeutics[J]. science, 2002, 297(5580): 353-356.
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[4].Mattson M P, Partin J, Begley J G. Amyloid β-peptide induces apoptosis-related events in synapses and dendrites[J]. Brain research, 1998, 807(1-2): 167-176.

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[8].Lecanu, L., Greeson, J., & Papadopoulos, V. (2006). Beta-amyloid and oxidative stress jointly induce neuronal death, amyloid deposits, gliosis, and memory impairment in the rat brain. Pharmacology, 76(1), 19-33.

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