PI3K/Akt/CREB activator 1

Cat. No.: HY-151527

CAS No.: 2708177-73-3

Molecular Formula: $C_{19}H_{15}F_4NO_3$

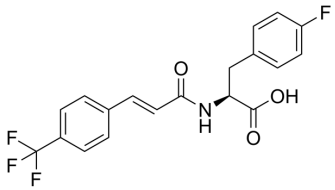
Molecular Weight: 381.32

Target: Akt; PI3K; Epigenetic Reader Domain

Pathway: PI3K/Akt/mTOR; Epigenetics

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (655.62 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6225 mL	13.1123 mL	26.2247 mL
5 mM	0.5245 mL	2.6225 mL	5.2449 mL
10 mM	0.2622 mL	1.3112 mL	2.6225 mL

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: ≥ 2.08 mg/mL (5.45 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.45 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PI3K/Akt/CREB activator 1 (compound AE-18) is a potent, orally active PI3K/Akt/CREB activator. PI3K/Akt/CREB activator 1 promotes neuronal proliferation, induced differentiation of Neuro-2a cells into a neuron-like morphology, and accelerated the establishment of axon-dendrite polarization of primary hippocampal neurons through upregulating brain-derived neurotrophic factor via the PI3K/Akt/CREB pathway. PI3K/Akt/CREB activator 1 can be used in research of vascular dementia (VaD)[1].

In Vitro

PI3K/Akt/CREB activator 1 (compound AE-18; 10 and 20 μM; 48 h) induces neurite outgrowth and proliferation through upregulating BDNF via the PI3K/Akt/CREB pathway Neuro-2a cells[1].
PI3K/Akt/CREB activator 1 (10 and 20 μM; neurons) enhances neuronal differentiation and axon-dendrite polarization in cultured hippocampal neurons through the PI3K/AKT signal pathway[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Neuro-2a cells
Concentration:	10 and 20 μM
Incubation Time:	48 hours
Result:	Increased the expressions of BDNF and the phosphorylated form of AKT (pAKT) and CREB (pCREB).

In Vivo

PI3K/Akt/CREB activator 1 (compound AE-18; 5 and 10 mg/kg; i.g.; male Sprague-Dawley rats with chronic cerebral hypoperfusion (CCH) model) improves cerebral blood flow (CBF) recovery after bilateral common carotid artery occlusion (BCCAO)[1].

PI3K/Akt/CREB activator 1 (5 and 10 mg/kg; i.g.; for 5 d) mitigates impairment of learning and memory in chronic cerebral hypoperfusion (CCH) rat model and alleviates CCH-induced pathological injury in the hippocampus after BCCAO[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (200-220 g) with chronic cerebral hypoperfusion (CCH) model[1]
Dosage:	5 and 10 mg/kg
Administration:	Oral gavage; daily, for 6 weeks
Result:	Promoted the recovery of CBF after BCCAO.

Animal Model:	Male Sprague-Dawley rats (200-220 g) with chronic cerebral hypoperfusion (CCH) model[1]
Dosage:	5 and 10 mg/kg
Administration:	Oral gavage; daily, for 5 days
Result:	Reduced escape latency from day 1 to day 5 of the morris water maze (MWM) test compared with the CCH group. Improved cognitive deficits in CCH rat model.

REFERENCES

[1]. Feng JH, et, al. Protective Effects of 4-Trifluoromethyl-(E)-cinnamoyl]-L-4-F-phenylalanine Acid against Chronic Cerebral Hypoperfusion Injury through Promoting Brain-Derived Neurotrophic Factor-Mediated Neurogenesis. ACS Chem Neurosci. 2022 Oct 16.

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