AP39

Cat. No.: HY-126124

CAS No.: 1429061-80-2

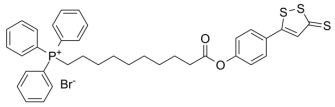
Molecular Formula: $C_{37}H_{38}BrO_2PS_3$

Molecular Weight: 721.77

Target: Others

Pathway: Others

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (138.55 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.3855 mL	6.9274 mL	13.8548 mL
	5 mM	0.2771 mL	1.3855 mL	2.7710 mL
	10 mM	0.1385 mL	0.6927 mL	1.3855 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: ≥ 0.83 mg/mL (1.15 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.83 mg/mL (1.15 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AP39 is a triphenylphosphonium derivatised anethole dithiolethione and mitochondria-targeting hydrogen sulfide (H_2S) donor. AP39 increases intracellular H_2S levels. AP39 exerts cytoprotective effects and maintains mitochondrial DNA integrity under oxidative stress conditions. AP39 protects against myocardial reperfusion injury in mice model and has the potential for Alzheimer's disease research[1][2][3].

In Vitro

AP39 (25,100 nM; for 24 h) results in increase in cell viability in APP/PS1 neurons and has no effect on cell viability in WT neurons[1].

AP39 (100 nM) increases the levels of OPA1 and Mfn1 but not Mfn2. Moreover, AP39 decreases the levels of Fis1 but not Drp1[1].

AP39 (25-250 nM; 2 h) induces a concentration-dependent increase in H_2S generation and in the fluorescence of the H_2S-detecting dye AzMC[1].

AP39 (100 nM) significantly increases the basal respiratory rate and the OCR-linked maximal respiratory capacity of the APP/PS1 neurons. AP39 significantly increases the ATP production in WT and APP/PS1 neurons. AP39 significantly protects against mtDNA damage in APP/PS1 neurons by partially restoring mtDNA integrity[1].
AP39 consists of a mitochondria-targeting motif, triphenylphosphonium (TPP^+), coupled to a H_2S-donating moiety (dithiolethione) by an aliphatic linker.AP39 (100 nM) reduces intracellular oxidative stress and in the meantime it consequently sustaines the cell viability, mitochondrial respiration and mitochondrial DNA integrity. These effects tend to be stimulatory at lower concentrations (30 and 100 nM), but tend to diminish or convert into inhibitory effects at a higher concentration (300 nM)[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

AP39 (100 nM/kg/day; ip; for 6 weeks) ameliorates the learning and memory deficits of APP/PS1 mice. AP39 (25-250 nM/kg/day; ip; for 6 weeks) induces a dose-dependent increase in H_2S generation in the cortex and hippocampus of WT and APP/PS1 mice[1].
AP39 (0.01, 0.1, 1 μmol/kg; iv; bolus 10 min before reperfusion) dose‐dependently reduces infarct size anaesthetized by thiobutabarbital (200 mg/kg, i.p) in male Sprague Dawley rats, 300-350 g (9-11 weeks)[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	12-month-old WT or APP/PS1 mice[1]
Dosage:	100 nM/kg
Administration:	IP; daily; for 6 weeks
Result:	Reversed the spatial learning and memory deficits of the aged AD model mice. Alleviated brain atrophy and ventricle asymmetryand inhibited Aβ plaque deposition in the brains in AD model mice.

CUSTOMER VALIDATION

- Antioxid Redox Signal. 2021 Aug 19.

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REFERENCES

[1]. Feng-Li Zhao, et al. AP39, a Mitochondria-Targeted Hydrogen Sulfide Donor, Supports Cellular Bioenergetics and Protects against Alzheimer's Disease by Preserving Mitochondrial Function in APP/PS1 Mice and Neurons. Oxid Med Cell Longev. 2016;2016:8360738.

[2]. Bartosz Szczesny, et al. AP39, a novel mitochondria-targeted hydrogen sulfide donor, stimulates cellular bioenergetics, exerts cytoprotective effects and protects against the loss of mitochondrial DNA integrity in oxidatively stressed endothelial cells in

[3]. Qutuba G Karwi, et al. AP39, a mitochondria-targeting hydrogen sulfide (H 2 S) donor, protects against myocardial reperfusion injury independently of salvage kinase signaling. Br J Pharmacol. 2017 Feb;174(4):287-301.

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