BAPTA-AM

Cat. No.: HY-100545

CAS No.: 126150-97-8

Molecular Formula: $C_{34}H_{40}N_2O_{18}$

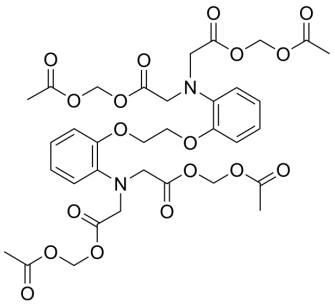
Molecular Weight: 764.68

Target: Potassium Channel

Pathway: Membrane Transporter/Ion Channel

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (65.39 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.3077 mL	6.5387 mL	13.0774 mL
5 mM	0.2615 mL	1.3077 mL	2.6155 mL
10 mM	0.1308 mL	0.6539 mL	1.3077 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: ≥ 2.5 mg/mL (3.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (3.27 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BAPTA-AM is a well-known membrane permeable Ca^{2+} chelator. BAPTA-AM inhibits hERG channels, hKv1.3 and hKv1.5 channels in HEK 293 cells with IC_{50}s of 1.3 μM, 1.45 μM and 1.23 μM, respectively[1].

IC_{50} & Target

Ca^{2+} chelator[1]

IC50: 1.3 μM (hERG channel, in HEK 293 cells), 1.45 μM (hKv1.3, in HEK 293 cells), 1.23 μM (hKv1.5, in HEK 293 cells)[1]

In Vitro	BAPTA-AM inhibits neuronal Ca^{2+}-activated K^+ channel currents, and up-regulates the decreased cardiac sodium current (INa) density by chelating intracellular Ca^{2+}[1]. BAPTA-AM (BAPTA/AM), an intracellular calcium chelator, induces delayed necrosis by lipoxygenase-mediated free radicals in mouse cortical cultures. BAPTA-AM prevents free radical-mediated toxicity promote apoptosis in non-neuronal cells and produce a beneficial effect in neuronal cells by protecting neurons from ischemic damage. In addition, it has been suggested that BAPTA-AM induces a late, but not early, increase of intracellular calcium in I-IL-60 neoplastic cells. Mixed cortical cell cultures (DIV 13-16) exposed to 10 μM BAPTA-AM for 24- or 48-hr show moderate (45-70%) neuronal injury as evaluated by increased LDH release into the bathing medium after 24-48-hr. Exposure of cortical cultures to 3-10 μM BAPTA-AM for 48-hr evoke dose-dependent neuronal damage[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Neuronal injury is quantitatively estimated by measuring lactate dehydrogenase (LDH) released from damaged cells into the bathing medium 24- or 48-hr after the 10 μM BAPTA/AM treatment. The morphological findings are confirmed by staining with neuron-specific enolase (NSE) antibody and tryphan blue[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2022 Feb 16;7(1):46.
- Nat Immunol. 2019 Apr;20(4):433-446.
- Cell Stem Cell. 2022 Oct 12;S1934-5909(22)00417-9.
- Nat Commun. 2023 Feb 6;14(1):642.
- Nat Commun. 2021 May 18;12(1):2915.

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REFERENCES

[1]. Wie MB, et al. BAPTA/AM, an intracellular calcium chelator, induces delayed necrosis by lipoxygenase-mediated free radicals in mouse cortical cultures. Prog Neuropsychopharmacol Biol Psychiatry. 2001 Nov;25(8):1641-59.

[2]. Tang Q, et al. The membrane permeable calcium chelator BAPTA-AM directly blocks human ether a-go-go-related gene potassium channels stably expressed in HEK 293 cells. Biochem Pharmacol. 2007 Dec 3;74(11):1596-607.

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