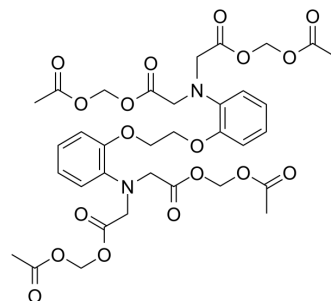


## BAPTA-AM

<b>Cat. No.:</b>	HY-100545		
<b>CAS No.:</b>	126150-97-8		
<b>Molecular Formula:</b>	C <sub>34</sub> H <sub>40</sub> N <sub>2</sub> O <sub>18</sub>		
<b>Molecular Weight:</b>	764.68		
<b>Target:</b>	Potassium Channel		
<b>Pathway:</b>	Membrane Transporter/Ion Channel		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : 50 mg/mL (65.39 mM; Need ultrasonic)  
 H<sub>2</sub>O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- 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
- 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
- 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<sup>2+</sup> chelator. BAPTA-AM inhibits hERG channels, hKv1.3 and hKv1.5 channels in HEK 293 cells with IC<sub>50</sub>s of 1.3 μM, 1.45 μM and 1.23 μM, respectively<sup>[1]</sup>.

#### IC<sub>50</sub> & Target

Ca<sup>2+</sup> chelator<sup>[1]</sup>  
 IC<sub>50</sub>: 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)<sup>[1]</sup>

## In Vitro

BAPTA-AM inhibits neuronal  $\text{Ca}^{2+}$ -activated  $\text{K}^+$  channel currents, and up-regulates the decreased cardiac sodium current (INa) density by chelating intracellular  $\text{Ca}^{2+}$ [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  $\mu\text{M}$  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  $\mu\text{M}$  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  $\mu\text{M}$  BAPTA/AM treatment. The morphological findings are confirmed by staining with neuron-specific enolase (NSE) antibody and trypan blue[1].

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

## CUSTOMER VALIDATION

- Nat Immunol. 2019 Apr;20(4):433-446.
- Signal Transduct Target Ther. 2022 Feb 16;7(1):46.
- Nat Commun. 2021 May 18;12(1):2915.
- Bone Res. 2022 Mar 8;10(1):25.
- Autophagy. 2021 Apr;17(4):925-947.

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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.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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