BQCA

Cat. No.: HY-101858

CAS No.: 338747-41-4

Molecular Formula: $C_{18}H_{15}NO_4$

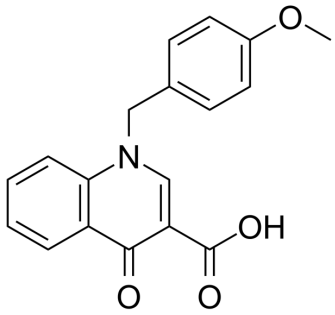
Molecular Weight: 309.32

Target: mAChR

Pathway: GPCR/G Protein; Neuronal Signaling

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 : 4.17 mg/mL (13.48 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2329 mL	16.1645 mL	32.3290 mL
	5 mM	0.6466 mL	3.2329 mL	6.4658 mL
	10 mM	0.3233 mL	1.6164 mL	3.2329 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

BQCA a highly selective allosteric modulator of the M1 mAChR.

In Vitro

BQCA reduces the concentration of ACh required to activate M1 up to 129-fold with an inflection point value of 845 nM. No potentiation, agonism, or antagonism activity on other mAChRs is observed up to 100 μM[1]. BQCA increases M1 receptor affinity for acetylcholine. The activation of the M1 receptor by BQCA induces a robust inward current and increases spontaneous excitatory postsynaptic currents in medial prefrontal cortex (mPFC) pyramidal cells[2].

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

In Vivo

BQCA requires M1 to promote inositol phosphate turnover in primary neurons and to increase c-fos and arc RNA expression and ERK phosphorylation in the brain. BQCA reverses scopolamine-induced memory deficits in contextual fear conditioning, increases blood flow to the cerebral cortex, and increases wakefulness while reducing delta sleep. BQCA induces β-arrestin recruitment to M1, suggesting a role for this signal transduction mechanism in the cholinergic modulation of memory[1]. BQCA increases firing of mPFC pyramidal cells in vivo. BQCA also restores discrimination reversal learning in a transgenic mouse model of Alzheimer's disease[2].

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PROTOCOL

Kinase Assay [1]

Competition binding reactions used 25 μg human M1 CHO membrane protein, BQCA or vehicle, and 0.15 nM [^{3}H]NMS in 96-well deep-well plates. Binding reactions (30 °C for 2-3 h) are terminated by rapid filtration. Nonspecific binding is determined by adding 10 μM atropine. Filter plates are ished 4×with ice-cold 20 mM HEPES, 100 mM NaCl, and 5 mM $MgCl_2$, pH 7.4 using a 96-well harvester. Plates are dried and radioactivity counted with a microplate scintillation counter[1].

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Animal Administration [1][2]

Rats: Male Sprague-Dawley rats weighing 225-250 g, are injected i.p. with the micro-suspension (containing 10% tween 80) of BQCA at the dose of 10 mg/kg. The blood and whole brain tissue samples are collected at 0.5, 1, 2, 4 and 8 h. Blood samples are collected through cardiac puncture in EDTA vacutainer tubes. The plasma is separated by centrifugation and stored at −80°C until analysis. The animals are decapitated and the whole brain tissue are removed and immediately frozen on dry ice[2].

Mice: Mice are dosed I.P. with BQCA in 5% beta-cyclodextrin and/or 0.3 mg/kg scopolamine in 0.9% saline 30 min before placement into a chamber for 2 min before 2 tone-footshock pairings (3 kHz, 85 dB tone for 30 s co-terminated with a 0.5 mA, 1 s shock) 2 min apart. Mice are removed to their home cage 30 s after the last pairing. Twenty-four hours later mice are placed into the same chamber and freezing is measured by Video Freeze[1].

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

REFERENCES

[1]. Ma L, et al. Selective activation of the M1 muscarinic acetylcholine receptor achieved by allosteric potentiation. Proc Natl Acad Sci U S A. 2009 Sep 15;106(37):15950-5.

[2]. Shirey JK, et al. A selective allosteric potentiator of the M1 muscarinic acetylcholine receptorincreases activity of medial prefrontal cortical neurons and restores impairments in reversal learning. J Neurosci. 2009 Nov 11;29(45):14271-86.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA