

Product Data Sheet

Product Name: Bay 60-7550

Cat. No.: GC19512

Chemical Properties

Cas. No. 439083-90-6

SMILES O=C1N=C(NN2C1=C(N=C2[C@@H](CCCC3=CC=CC=C3)[C@@H](C)O)C)CC4=CC=C(C(OC)=C4)OC

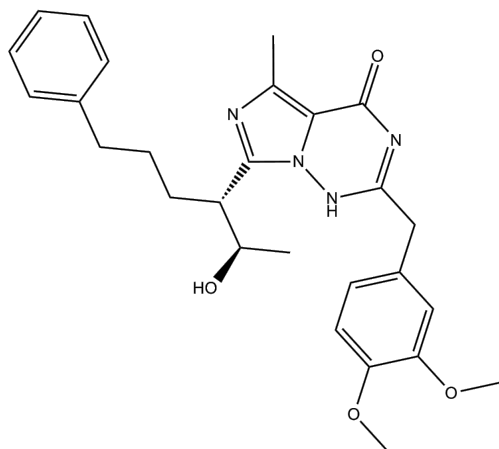
Formula $C_{27}H_{32}N_4O_4$ M.Wt 476.57

Solubility Acetone: 10 mg/ml, DMSO: 10 mg/ml, Ethanol: 10 mg/ml Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines HT-22 cells

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

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HT-22 cells were cultured in DMEM medium containing 10% FBS at 37°C in 5% CO₂. Cells were seeded in 96-well plates at a density of 1×10^5 cells/well. Cells were divided into three groups: control group, 50μM corticosterone treatment Preparation Method group, and 50μM corticosterone+1μM Bay 60-7550 experimental group. After 24 hours, 10μl MTT solution was added to each well and incubated for an additional 4 hours at 37°C. The medium in each well was then replaced with 200μl DMSO, and the absorbance at 570nm was measured.

Reaction
Conditions 1μM; 24h

Applications Bay 60-7550 treatment significantly rescued HT-22 cells from corticosterone-induced cytotoxicity.

Animal experiment [2]:

Animal models Male C57bL/6 mice

Male C57bL/6 mice (25-30g; 3 months of age) were housed at a controlled temperature (22±1°C) with a 12h/12h light/dark cycle (lights on at 7:00 am). The Preparation Method mice had ad libitum access to food and water. Transient cerebral ischemia was induced by BCCAO. Vehicle or BAY 60-7550 was administered by oral gavage (p.o.) at a dose of 1mg/kg/day for 21 days, followed by behavioral testing.

Dosage form 1mg/kg/day for 21 days; p.o.

Applications BAY 60-7550 treatment alleviated BCCAO-induced behavioral disorders and anxiety in mice.

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References:

- [1] Xu Y, Pan J, Chen L, et al. Phosphodiesterase-2 inhibitor reverses corticosterone-induced neurotoxicity and related behavioural changes via cGMP/PKG dependent pathway[J]. International Journal of Neuropsychopharmacology, 2013, 16(4): 835-847.
- [2] Soares L M, Meyer E, Milani H, et al. The phosphodiesterase type 2 inhibitor BAY 607550 reverses functional impairments induced by brain ischemia by decreasing hippocampal neurodegeneration and enhancing hippocampal neuronal plasticity[J]. European Journal of Neuroscience, 2017, 45(4): 510-520.

Background

Bay 60-7550 is a potent inhibitor of Phosphodiesterase 2 (PDE2) purified from bovine heart with an IC_{50} value of $2.0 \pm 0.7 nM$ ^[1]. Bay 60-7550 increased cGMP and cAMP levels and promoted cGMP signaling^[2]. Bay 60-7550 has been widely used in different animal models to improve depression-like behaviors and enhance brain function^[3].

In vitro, Bay 60-7550 treatment ($1 \mu M$) for 24h rescued HT-22 cells from the cytotoxicity induced by $50 \mu M$ corticosterone and reversed down-regulation of CBP proteins induced by corticosterone^[4]. Treatment with $1 \mu M$ Bay 60-7550 for 48 hours blocked oxidative stress and apoptosis in mouse primary cortical neuronal cells treated with $A\beta_{1-42}$ oligomers^[5].

In vivo, Bay 60-7550 treatment via intraperitoneal injection at a dose of 3mg/kg twice daily for 4 days reduced ethanol preference and intake without affecting locomotor activity in C57BL/6J mice^[6]. BAY 60-7550 treatment (p.o.) at a daily dose of 1mg/kg for 21 days alleviated bilateral common carotid artery occlusion (BCCAO)-induced behavioral deficits in mice and enhanced the expression of pCREB and BDNF proteins in the hippocampus of mice^[7]. Intraperitoneal injection of BAY 60-7550 at a daily dose of 3mg/kg for 14 days reversed $A\beta_{1-42}$ -induced memory impairment in mice^[8].

References:

- [1] Boess F G, Hendrix M, van der Staay F J, et al. Inhibition of phosphodiesterase 2 increases neuronal cGMP, synaptic plasticity and memory performance[J]. Neuropharmacology, 2004, 47(7): 1081-1092.

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- [2] Masood A, Huang Y, Hajjhussein H, et al. Anxiolytic effects of phosphodiesterase-2 inhibitors associated with increased cGMP signaling[J]. The Journal of pharmacology and experimental therapeutics, 2009, 331(2): 690-699.
- [3] Huang X, Xiaokaiti Y, Yang J, et al. Inhibition of phosphodiesterase 2 reverses gp91phox oxidase-mediated depression-and anxiety-like behavior[J]. Neuropharmacology, 2018, 143: 176-185.
- [4] Xu Y, Pan J, Chen L, et al. Phosphodiesterase-2 inhibitor reverses corticosterone-induced neurotoxicity and related behavioural changes via cGMP/PKG dependent pathway[J]. International Journal of Neuropsychopharmacology, 2013, 16(4): 835-847.
- [5] Yan Y, Gao S, Avasthi S, et al. Protective effects of phosphodiesterase 2 inhibitor against A β 1-42 induced neuronal toxicity[J]. Neuropharmacology, 2022, 213: 109128.
- [6] Shi J, Liu H, Pan J, et al. Inhibition of phosphodiesterase 2 by Bay 60-7550 decreases ethanol intake and preference in mice[J]. Psychopharmacology, 2018, 235(8): 2377-2385.
- [7] Soares L M, Meyer E, Milani H, et al. The phosphodiesterase type 2 inhibitor BAY 60-7550 reverses functional impairments induced by brain ischemia by decreasing hippocampal neurodegeneration and enhancing hippocampal neuronal plasticity[J]. European Journal of Neuroscience, 2017, 45(4): 510-520.
- [8] Ruan L, Du K, Tao M, et al. Phosphodiesterase-2 inhibitor bay 60-7550 ameliorates A β -induced cognitive and memory impairment via regulation of the HPA Axis[J]. Frontiers in cellular neuroscience, 2019, 13: 432.

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