

Product Data Sheet

Product Name: Butylphthalide (3-n-Butylphthalide)

Cat. No.: GN10524

Chemical Properties

Cas. No. 6066-49-5

Chemical Name 3-butyl-3H-2-benzofuran-1-one

SMILES CCCCC1C2=CC=CC=C2C(=O)O1

Formula $C_{12}H_{14}O_2$

M.Wt 190.24

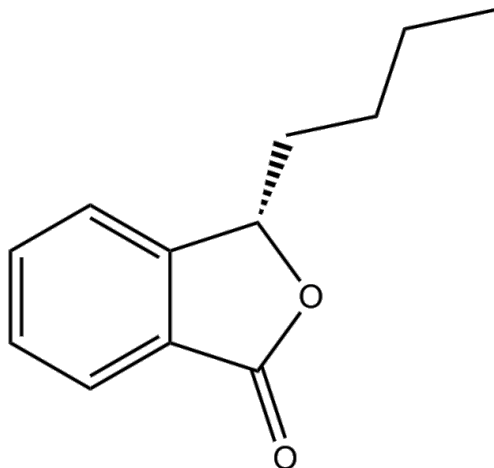
Solubility $\geq 19\text{mg/mL}$ in DMSO

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 SV40-transformed aortic rat endothelial cell line (SVAREC)

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

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Preparation Method SVAREC cells were cultured in Roswell Park Memorial Institute-1640 (RPMI-1640) medium supplemented with 10% heat-inactivated fetal bovine serum (FBS) at 37°C, 5% CO₂, and 95% air. Cells were subjected to oxygen-glucose deprivation (OGD) for 6 hours in the presence of 1μM Butylphthalide.

Reaction Conditions 1μM; 6h

Applications Butylphthalide protected eNOS activity during OGD. Butylphthalide treatment significantly alleviated OGD-induced cell injury, improving cell viability and morphology.

Animal experiment [2]:

Animal models C57BL/6 mice

Preparation Method Mice were intragastrically administered rotenone for 8 consecutive weeks (0.25μmol/L) to induce Parkinson's disease (PD) symptoms. Butylphthalide (200mg/kg/day) was administered intraperitoneally for 6 weeks (from the 5th to the 10th week) to observe its protective effects.

Dosage form 200mg/kg; i.p.

Applications Butylphthalide alleviated behavioral impairments in rotenone-induced PD mice, protected against dopaminergic neuron loss, inhibited microglial activation, and reduced neuroinflammation by inhibiting the cGAS-STING pathway.

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- [1] Wei H, Zhan LP, Zhang B, et al. dl-3n-butylphthalide reduces oxygen-glucose deprivation-induced endothelial cell damage by increasing PGC-1 α . Eur Rev Med Pharmacol Sci. 2019 May;23(10):4481-4490.
- [2] Liu Y, Duan R, Li P, et al. 3-N-butylphthalide attenuates neuroinflammation in rotenone-induced Parkinson's disease models via the cGAS-STING pathway. Int J Immunopathol Pharmacol. 2024 Jan-Dec;38:3946320241229041.

Background

Butylphthalide (3-n-Butylphthalide) is a natural compound with a wide range of pharmacological activities, primarily used for the treatment of acute ischemic stroke^[1]. The mechanisms of action of Butylphthalide include antioxidant effects and mitochondrial protection^[2]. Butylphthalide can also improve symptoms of myocardial infarction^[3]. In addition, Butylphthalide has the potential to treat tissue damage induced by ischemia-reperfusion^[4].

In vitro, pretreatment of SV40-transformed rat aortic endothelial cells with Butylphthalide (1 μ M) for 6 hours, followed by oxygen-glucose deprivation (OGD) for 6 hours, significantly enhances PGC-1 α expression and protects cells from OGD-induced damage, increasing cell viability^[5]. Pretreatment of SH-SY5Y cells with Butylphthalide (5 μ M) for 6 hours, followed by stimulation with 6-OHDA (100 μ M) for 24 hours, significantly increases cell viability, reduces cell apoptosis and ROS production, inhibits NLRP3 inflammasome activation and related inflammatory factor expression, alleviates mitochondrial damage, and decreases abnormal aggregation of α -synuclein (α -Syn)^[6].

In vivo, Butylphthalide (200mg/kg) is administered intraperitoneally once daily to C57BL/6 mice starting at 8 weeks of age until the 10th week. Butylphthalide significantly alleviates behavioral deficits induced by Rotenone, reduces the loss of dopaminergic neurons, and inhibits microglial activation^[7]. Butylphthalide (4mg/kg) is administered intraperitoneally as a single dose to rats with middle cerebral artery occlusion/reperfusion (MCAO/R) model. Butylphthalide significantly alleviates behavioral deficits induced by MCAO/R, reduces brain infarct volume, inhibits microglial activation, suppresses ferroptosis by activating the Nrf2/HO-1 signaling pathway, and alleviates lipid peroxidation, iron accumulation, and mitochondrial damage induced by ischemia-reperfusion^[8].

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- [1] Chen XQ, Qiu K, Liu H, et al. Application and prospects of butylphthalide for the treatment of neurologic diseases. Chin Med J (Engl). 2019 Jun 20;132(12):1467-1477.
- [2] Zhu T, Wang L, Feng Y, et al. Classical Active Ingredients and Extracts of Chinese Herbal Medicines: Pharmacokinetics, Pharmacodynamics, and Molecular Mechanisms for Ischemic Stroke. Oxid Med Cell Longev. 2021 Mar 13;2021:8868941.
- [3] Bai M, Pan CL, Jiang GX, et al. Effects of butylphthalide on oxidative stress and inflammatory response in rats with myocardial infarction through Akt/Nrf2 signaling pathway. Eur Rev Med Pharmacol Sci. 2019 Nov;23(21):9642-9650.
- [4] Lu P, Li WP, Zhou BJ, et al. N-butylphthalide (NBP) ameliorated ischemia/reperfusion-induced skeletal muscle injury in male mice via activating Sirt1/Nrf2 signaling pathway. Physiol Rep. 2024 Dec;12(23):e70149.
- [5] Wei H, Zhan LP, Zhang B, et al. dl-3n-butylphthalide reduces oxygen-glucose deprivation-induced endothelial cell damage by increasing PGC-1 α . Eur Rev Med Pharmacol Sci. 2019 May;23(10):4481-4490.
- [6] Que R, Zheng J, Chang Z, et al. DI-3-n-Butylphthalide Rescues Dopaminergic Neurons in Parkinson's Disease Models by Inhibiting the NLRP3 Inflammasome and Ameliorating Mitochondrial Impairment. Front Immunol. 2021 Dec 1;12:794770.
- [7] Liu Y, Duan R, Li P, et al. 3-N-butylphthalide attenuates neuroinflammation in rotenone-induced Parkinson's disease models via the cGAS-STING pathway. Int J Immunopathol Pharmacol. 2024 Jan-Dec;38:3946320241229041.
- [8] Sun M, Chen J, Liu F, et al. Butylphthalide inhibits ferroptosis and ameliorates cerebral Ischaemia-Reperfusion injury in rats by activating the Nrf2/HO-1 signalling pathway. Neurotherapeutics. 2024 Sep;21(5):e00444.

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