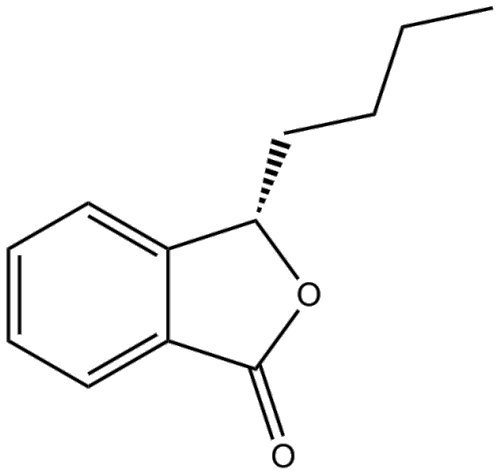
Product Data Sheet

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	≥ 19mg/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)

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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% CO_2, 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.

Caution: Product has not been fully validated for medical applications. For research use only.
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References:
[1] Wei H, Zhan LP, Zhang B, et al. dl-3n-butylphthalide reduces oxygen-glucose deprivation-induced endothelial cell damage by increasing PGC-1α. 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 (1μM) for 6 hours, followed by oxygen-glucose deprivation (OGD) for 6 hours, significantly enhances PGC-1α expression and protects cells from OGD-induced damage, increasing cell viability[5]. Pretreatment of SH-SY5Y cells with Butylphthalide (5μM) for 6 hours, followed by stimulation with 6-OHDA (100μM) 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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References:

[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-1α. Eur Rev Med Pharmacol Sci. 2019 May;23(10):4481-4490.
[6] Que R, Zheng J, Chang Z, et al. Dl-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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