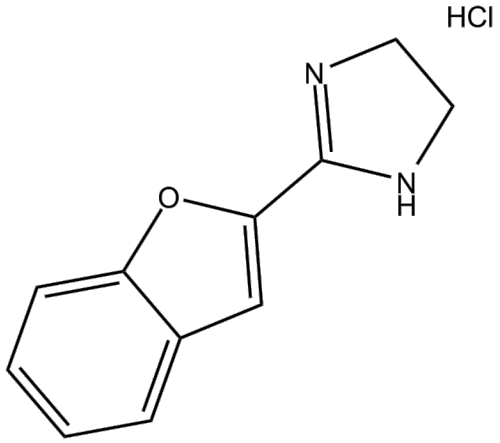
Product Data Sheet

Product Name: 2-BFI hydrochloride
Cat. No.: GC11588

Chemical Properties

Cas. No.	89196-95-2		
Chemical Name	2-(benzofuran-2-yl)-4,5-dihydro-1H-imidazole hydrochloride		
SMILES	C12=CC=CC=C1OC(C3=NCCN3)=C2.Cl		
Formula	C11H11ClN2O	M.Wt	222.67
Solubility	22.2mg/ml in Water	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: Human colorectal adenocarcinoma cell lines HT-29 and HCT-116

Product Data Sheet

Preparation Method	Human colorectal adenocarcinoma cell lines HT-29 and HCT-116 were cultured in McCoy's 5A Medium (modified), with L-Glutamine, supplemented with 10% fetal bovine serum, 100units/mL penicillin and 100μg/mL streptomycin. Cell cultures were incubated in a humidified atmosphere of 95% air and 5% CO_2 at 37°C. 2-BFI hydrochloride was dissolved in water as a 10mM masterbatch and storage at –80°C refrigerator. HT-29 and HCT-116 cells were seeded in triplicate on 96-well plates at a density of 10×10^3cells/100μL. The next day, cells were exposed to 2-BFI (250μM) and/or HCQ for 24h and 48h. The MTT assay was used to measure cell viability according to the commercially available protocol. The generation of intracellular ROS level and apoptosis detection were measured using flow cytometry technique. The Autophagy Kit was used to monitor autophagic flux.
Reaction Conditions	250μM; 24-48h
Applications	Treatment of HT-29 and HCT-116 colorectal cancer cells with 2-BFI hydrochloride significantly potentiated hydroxychloroquine cytotoxicity, elevated ROS, increased early/late apoptosis, and blocked autophagic flux.

Animal experiment [2]:

Animal models	Male adult Sprague-Dawley rats

Product Data Sheet

Preparation Method	Male adult (2-month-old, 250-300g) Sprague-Dawley rats were housed in a temperature (23±1°C) and humidity (50%) controlled animal facility with 12:12h light–dark cycle. Food and water were available ad libitum. Focal cerebral ischemic stroke model was established by permanent distal middle cerebral artery occlusion (dMCAO) method. Immediately following dMCAO, rats were randomly selected and administered 2-BFI hydrochloride (3mg/kg) or saline intraperitoneally. Rats undergo neurobehavioral tests to evaluate functional outcome 1 and 3 days after ischemia. The modified beam balance test was used to assess the motor balance and coordination in ischemic rats, especially in the hindlimb. The brains of rat were removed 3 days after ischemia, 2mm coronal sections were cut and stained with 2,3,5-triphenyltetrazolium hydrochloride (TTC). Western blotting procedures were performed to assess the profile of mTOR activation. To test the inflammatory response after 2BFI treatment, colorimetric ELISA kits were used to detect the following cytokines in brain protein extract: IL-10,TNF-α, TGF-β.
Dosage form	3mg/kg; i.p.; 3 days
Applications	2-BFI hydrochloride reduced cerebral infarct volume, improved motor deficits in dMCAO rats, inhibited mTOR phosphorylation and shifted cytokine balance toward anti-inflammation.

References:

[1] Kowalski S, Wityk P, Raczak-Gutknecht J, Olszewska A, Zmijewski M, Kocic I. The imidazoline I_2 receptor agonist 2-BFI enhances cytotoxic activity of hydroxychloroquine by modulating oxidative stress, energy-related metabolism and autophagic influx in human colorectal adenocarcinoma cell lines. *Eur J Pharmacol*. 2025;996:177590.
[2] Cheng Y, Zhang W, Cao W, et al. 2-BFI attenuates ischemic injury by modulating mTOR signaling and neuroinflammation in rats. *Neurosci Lett*. 2021;750:135766.

Background

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Background

2-BFI hydrochloride is a high-affinity, selective agonist of the imidazoline I2 receptor (I2R) with a Ki of 70.1nM[1]. I2R is an allosteric binding site on monoamine oxidase-B that modulates neurotransmitter metabolism and neuroprotection[2]. 2-BFI hydrochloride is commonly used in studies of neuroprotection, anti-inflammation, and traumatic brain injury (TBI)[3].

In vitro, treatment of cultured rat cortical neurons with 2-BFI hydrochloride (30-1000μM; 2-3min) selectively inhibits NMDA-evoked currents[4]. Treatment of HT-29 and HCT-116 colorectal cancer cells with 2-BFI hydrochloride (250μM; 24-48h) significantly potentiated hydroxychloroquine cytotoxicity, elevated ROS, increased early/late apoptosis, and blocked autophagic flux[5].

In vivo, 2-BFI hydrochloride (3mg/kg; i.p.; 3 days) reduced cerebral infarct volume, improved motor deficits in distal middle cerebral artery occlusion (dMCAO) rats, inhibited mTOR phosphorylation and shifted cytokine balance toward anti-inflammation[6]. 2-BFI hydrochloride (10mg/kg; i.p.; twice daily for 7 days) reversed CCI-induced mechanical and thermal hypersensitivity, and reduced spinal Iba-1 and GFAP expression and TNF-α levels in neuropathic pain rats[7].

References:

[1] Carpéné C, Collon P, Remaury A, et al. Inhibition of amine oxidase activity by derivatives that recognize imidazoline I2 sites. *J Pharmacol Exp Ther*. 1995;272(2):681-688.
[2] Li JX. Imidazoline I_2 receptors: An update. *Pharmacol Ther*. 2017;178:48-56.
[3] Ni H, Rui Q, Lin X, Li D, Liu H, Chen G. 2-BFI Provides Neuroprotection Against Inflammation and Necroptosis in a Rat Model of Traumatic Brain Injury. *Front Neurosci*. 2019;13:674.
[4] Xu S, Chen J, Xu C, et al. 2-BFI protects against ischemic stroke by selectively acting on NR2B-containing NMDA receptors. *Brain Res*. 2024;1845:149284.
[5] Kowalski S, Wityk P, Raczak-Gutknecht J, Olszewska A, Zmijewski M, Kocic I. The imidazoline I_2 receptor agonist 2-BFI enhances cytotoxic activity of hydroxychloroquine by modulating oxidative stress, energy-related metabolism and autophagic influx in human colorectal adenocarcinoma cell lines. *Eur J Pharmacol*. 2025;996:177590.
[6] Cheng Y, Zhang W, Cao W, et al. 2-BFI attenuates ischemic injury by modulating mTOR signaling and neuroinflammation in rats. *Neurosci Lett*. 2021;750:135766.
[7] Siemian JN, LaMacchia ZM, Spreuer V, et al. The imidazoline I_2 receptor agonist 2-BFI

attenuates hypersensitivity and spinal neuroinflammation in a rat model of neuropathic pain. *Biochem Pharmacol*. 2018;153:260-268.