Benzbromarone

Cat. No.: HY-B1135

CAS No.: 3562-84-3

Molecular Formula: $C_{17}H_{12}Br_2O_3$

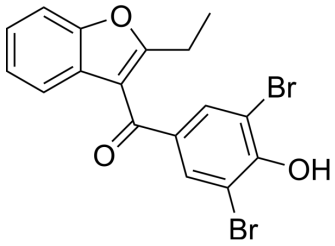
Molecular Weight: 424.08

Target: Xanthine Oxidase; Apoptosis; Interleukin Related; Keap1-Nrf2; SOD; Caspase; Bcl-2 Family; NF-κB; JNK; HSP

Pathway: Metabolic Enzyme/Protease; Apoptosis; Immunology/Inflammation; NF-κB; MAPK/ERK Pathway; Cell Cycle/DNA Damage

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (235.80 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3580 mL	11.7902 mL	23.5805 mL
	5 mM	0.4716 mL	2.3580 mL	4.7161 mL
	10 mM	0.2358 mL	1.1790 mL	2.3580 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.90 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (4.90 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Benzbromarone is an orally active anti-gout agent. Benzbromarone has anti-infammatory, anti-oxidative stress and nephroprotective effects. Benzbromarone can be used for the research of hyperuricemia and gout[1][2][3][4].

IC_{50} & Target				
	IL-1β	IL-8	Caspase-8	Caspase 9

	Caspase 3	Bcl-2

In Vitro

Benzbromarone (5-20 μM, 24 h) protects against propofol (HY-B0649)‐induced cytotoxicity in Human brain microvascular endothelial cells (HBMVECs)[1].
Benzbromarone (5-20 μM, 24 h) mitigates propofol (HY-B0649)‐induced oxidative stress and inhibits expression of pro‐inflammatory cytokines and Chemokines in HBMVECs[1].
Benzbromarone (1-100 μM, 2-24 h) activats the NRF2 signaling pathway in HepG2 cells[2].
Benzbromarone (1-30 μM, 24 h) promotes degradation of HSP47 to ameliorate collagen overproduction in human keloid fibroblasts[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cytotoxicity Assay[1]

Cell Line:	HBMVECs
Concentration:	5 μM, 10 μM, 20 μM
Incubation Time:	24 h
Result:	Improved the viability reduced by propofol.

Western Blot Analysis[2]

Cell Line:	HepG2 cells
Concentration:	1 μM, 2 μM, 5 μM, 10 μM, 20 μM, 50 μM, 100 μM
Incubation Time:	2 h, 6 h, 24 h
Result:	Increased NRF2 protein expression in HepG2 cells exposed for 2 h, 6 h and 24 h at any concentration. Significantly accumulated the protein of NRF2 in the nuclear fraction after exposure to 100 μM at any time point. Caused an increase in the protein expression of TRX1 and TRX2. Significantly increased the ratio of oxidized TRX2 to reduced TRX2 at a concentration of 100 μM.

In Vivo

Benzbromarone (25-50 mg/kg, Intragastric, once a day for four weeks) aggravates hepatic steatosis in high fat diet (HFD)-induced obese (DIO) mice[3].
Benzbromarone (10 mg/kg, Oral gavage, once a day for 14 consecutive days) attenuates the nephrotoxicity caused by cisplatin(HY-17394) in cisplatin treated rats[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	High fat diet (HFD)-induced obese (DIO) mice[3]
Dosage:	25 mg/kg, 50 mg/kg
Administration:	Intragastric (i.g.)
Result:	Aggravated lipid accumulation in the liver of DIO mice. Significantly increased triglyceride accumulation and AST, ALT levels. Regulated multiple lipid metabolism genes and the expression of protein markers associated with apoptosis, endoplasmic reticulum stress, and inflammation in the liver of DIO mice.

Animal Model:	Cisplatin treated rats[2]
Dosage:	10 mg/kg
Administration:	Oral gavage (p.o.)
Result:	Ameliorated the elevation in serum creatinine and blood urea nitrogen (BUN) levels induced by cisplatin. Counteracted oxidative stress induced by cisplatin and enhances anti-oxidant defenses in kidney. Alleviated the inflammatory events of nephrotoxicity induced by cisplatin. Attenuated cisplatin-induced apoptosis.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2023 Jun 21;e2300881.
- Stem Cell Res Ther. 2020 May 26;11(1):200.
- Biotechnol Bioeng. 2021 Sep 3.

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REFERENCES

[1]. Huang Z, et al. The protective effects of benzbromarone against propofol-induced inflammation and injury in human brain microvascular endothelial cells (HBMVECs) [J]. Neurotoxicity Research, 2021, 39(5): 1449-1458.

[2]. Roos N J, et al. The uricosuric benzbromarone disturbs the mitochondrial redox homeostasis and activates the NRF2 signaling pathway in HepG2 cells [J]. Free Radical Biology and Medicine, 2020, 152: 216-226.

[3]. Sun P, et al. Benzbromarone aggravates hepatic steatosis in obese individuals [J]. Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease, 2018, 1864(6): 2067-2077.

[4]. Abdel-Razek E A N, et al. Benzbromarone mitigates cisplatin nephrotoxicity involving enhanced peroxisome proliferator-activated receptor-alpha (PPAR-α) expression [J]. Life sciences, 2020, 243: 117272.

[5]. Park J G, et al. Benzbromarone Induces Targeted Degradation of HSP47 Protein and Improves Hypertrophic Scar Formation [J]. Journal of Investigative Dermatology, 2023.

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