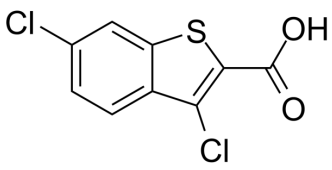
BT2

Cat. No.:	HY-114855
CAS No.:	34576-94-8
Molecular Formula:	$C_9H_4Cl_2O_2S$
Molecular Weight:	247.1
Target:	Bcl-2 Family
Pathway:	Apoptosis
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (252.93 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.0469 mL	20.2347 mL	40.4694 mL
	5 mM	0.8094 mL	4.0469 mL	8.0939 mL
	10 mM	0.4047 mL	2.0235 mL	4.0469 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 (8.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (8.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BT2 is a BCKDC kinase (BDK) inhibitor with an IC_{50} of 3.19 μM. BT2 binding to BDK triggers helix movements in the N-terminal domain, resulting in the dissociation of BDK from the branched-chain α-ketoacid dehydrogenase complex (BCKDC) [1]. BT2 (compound 4) is also a potent and selective Mcl-1 inhibitor with a K_i value of 59 μM[2].

IC_{50} & Target

BDK	Mcl-1
3.19 μM (IC_{50})	59 μM (Ki)

In Vivo

BT2 (20 mg/kg/day; intraperitoneal injection; daily; for 7 days; C57BL/6J male mice) treatment robustly enhances BCKDC activity in the heart (12.3-fold) compared with the vehicle-treated animals. Less activation is obtained in muscle and kidney at 3.6- and 3.8-fold, respectively. The -fold activation of BCKDC activity in the above tissues correlates with decreased phosphorylation in heart, muscle, and kidney after the long term BT2 treatment. BT2 treatment reduces the protein levels of

BDK in kidneys and heart[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6J male mice (8-10-week-old)[1]
Dosage:	20 mg/kg/day
Administration:	Intraperitoneal injection; daily; for 1 week
Result:	BCKDC activity was robustly (12.3-fold) enhanced in the heart compared with the vehicle-treated animals. Less activation was obtained in muscle and kidney at 3.6- and 3.8-fold, respectively. The protein levels of BDK in kidneys and heart were reduced to averages of 39 and 24%, respectively.

CUSTOMER VALIDATION

- Research Square Preprint. 2023 Jul 19.

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REFERENCES

[1]. Tso SC, et al. Benzothiophene carboxylate derivatives as novel allosteric inhibitors of branched-chain α-ketoacid dehydrogenase kinase. J Biol Chem. 2014 Jul 25;289(30):20583-93.

[2]. Friberg A, et al. Discovery of potent myeloid cell leukemia 1 (Mcl-1) inhibitors using fragment-based methods and structure-based design. J Med Chem. 2013 Jan 10;56(1):15-30.

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