BEC hydrochloride

Cat. No.: HY-19548A

CAS No.: 222638-67-7

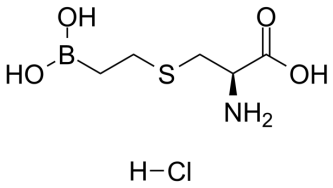
Molecular Formula: $C_5H_{13}BClNO_4S$

Molecular Weight: 229.49

Target: Arginase

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (217.87 mM; Need ultrasonic)
DMSO : 50 mg/mL (217.87 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.3575 mL	21.7874 mL	43.5749 mL
5 mM	0.8715 mL	4.3575 mL	8.7150 mL
10 mM	0.4357 mL	2.1787 mL	4.3575 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 140 mg/mL (610.05 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.89 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.89 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BEC hydrochloride is a slow-binding and competitive Arginase II inhibitor with K_i of 0.31 μM and 30 nM at pH 7.5 and pH 9.5, respectively[1].

IC_{50} & Target

Ki: 0.31 μM (BEC hydrochloride, at pH 7.5) and 30 nM (BEC hydrochloride, at pH 9.5)[1]

In Vitro

The X-ray crystal structure of the arginase-BEC complex has been determined at 2.3 Å resolution from crystals perfectly

twinned by hemihedry. The structure of the complex reveals that the boronic acid moiety undergoes nucleophilic attack by metal-bridging hydroxide ion to yield a tetrahedral boronate anion that bridges the binuclear manganese cluster, thereby mimicking the tetrahedral intermediate (and its flanking transition states) in the arginine hydrolysis reaction[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Administration of the arginase inhibitor BEC decreases arginase activity and causes alterations in NO homeostasis, which are reflected by increases in S-nitrosylated and nitrated proteins in the lungs from inflamed mice. BEC enhances perivascular and peribronchiolar lung inflammation, mucus metaplasia, NF-κB DNA binding, and mRNA expression of the NF-κB-driven chemokine genes CCL20 and KC, and leads to further increases in airways hyperresponsiveness[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6J wild-type mice, mice deficient in arginase 2 ($Arg2^{-/-}$), mice deficient in both arginase 1 and 2 ($Arg1^{-/-}Arg2^{-/-}$), and mice deficient in NOX2 ($NOX2^{-/-}$
Dosage:	20 mg/kg.
Administration:	I.V., in 0.9% saline, 1 hour before the injection of LPS.
Result:	BEC robustly reduced VEGF expression in neuroglia (72% reduction) and macrophage/microglia (87% reduction).

CUSTOMER VALIDATION

- Free Radic Biol Med. 2023 Feb 23;S0891-5849(23)00090-4.

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REFERENCES

[1]. Colleluori DM, et al. Classical and slow-binding inhibitors of human type II arginase. Biochemistry. 2001 Aug 7;40(31):9356-62.

[2]. Kim NN, et al. Probing erectile function: S-(2-boronoethyl)-L-cysteine binds to arginase as a transition state analogue and enhances smooth muscle relaxation in human penile corpus cavernosum. Biochemistry. 2001 Mar 6;40(9):2678-88.

[3]. Ckless K, et al. Inhibition of arginase activity enhances inflammation in mice with allergic airway disease, in association with increases in protein S-nitrosylation and tyrosine nitration. J Immunol. 2008 Sep 15;181(6):4255-64.

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