
Product Data Sheet

Product Name: BEC
Cat. No.: GC10280

Chemical Properties

Cas. No. 63107-40-4

Chemical Name S-(2-boronoethyl)-L-cysteine

SMILES OB(O)CCSC[C@@H](C(O)=O)N

Formula C₅H₁₂BNO₄S M.Wt 193.0

Solubility ≤5mg/ml in PBS(pH7.2) 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

Background

K(I): 0.4-0.6 microM

S-(2-boronoethyl)-L-cysteine (BEC) is an arginase inhibitor.

Arginases can catalyze the hydrolysis of L-arginine to yield L-ornithine and urea. Recently, studies show that arginases, both the type I and type II isozymes, involve in the regulation of nitric oxide production via modulating the availability of arginine for nitric oxide synthase.

In vitro: Although BEC has been first identified as inhibitor of type I arginase, it was found to be a classical, competitive inhibitor of human type II arginase with K(i) value 0.31 microM at pH 7.5. However, at pH 9.5, BEC was a slow-binding inhibitor of the enzyme with K(i) value 30 nM [1].

In vivo: In animal study, the administration of BEC was found to be able to decrease

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arginase activity and cause alterations in NO homeostasis, which were indicated by increases in S-nitrosylated and nitrated proteins in the lungs from inflamed mice. Moreover, in contrast to first expectations, BEC could enhance perivascular and peribronchiolar lung inflammation, NF- κ B DNA binding, mucus metaplasia, and mRNA expression of the NF- κ B-driven chemokine genes including KC and CCL20, and result in further increases in airways hyperresponsiveness [2].

Clinical trial: Up to now, BEC is still in the preclinical development stage.

References:

- [1] N. N. Kim, J. D. Cox, R. F. Baggio, 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* 40, 2678-2688 (2001).
- [2] 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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