

Product Data Sheet

Product Name: Bortezomib (PS-341)

Cat. No.: GC17644

Chemical Properties

Cas. No. 179324-69-7

Chemical Name [(1R)-3-methyl-1-[[[(2S)-3-phenyl-2-(pyrazine-2-carbonylamino)propanoyl]amino]butyl]boronic acid

SMILES B(C(CC(C)C)NC(=O)C(CC1=CC=CC=C1)NC(=O)C2=NC=CN=C2)(O)O

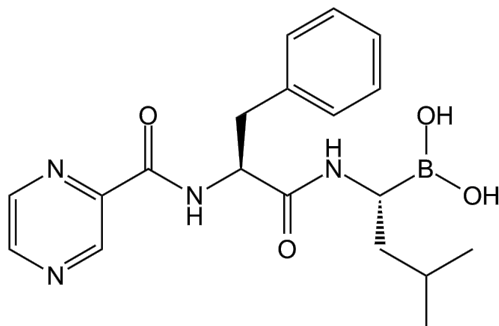
Formula $C_{19}H_{25}BN_4O_4$ M.Wt 384.24

Solubility $\geq 19.212\text{mg/mL}$ in DMSO Storage 4°C , protect from light

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 bone marrow (BM) cells

Caution: Product has not been fully validated for medical applications. For research use only.

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Proliferation inhibition analysis of DCs in bone marrow (BM) cells was treated Preparation Method with 10 nM - 50 nM Bortezomib for 24 hours, 48 hours and 72 hours, respectively.

Reaction Conditions 10 nM - 50 nM; for 24 hours, 48 hours and 72 hours

Applications The proliferation of DCs was significantly inhibited when treating with Bortezomib at 10 nM for 24 hours compared to control cells. The inhibition was significantly potentiated with higher concentrations of Bortezomib. Compared to untreated cells, less than 40% cells were detected when treated with Bortezomib at 50 nM for 24 hours. The inhibition of Bortezomib to DCs is both dose- and time-dependent.

Animal experiment [2]:

Animal models Six-week-old BALB/c female athymic nude mice

Preparation Method Five days after HCS-2/8 and OUMS-27 cells were injected subcutaneously into the right armpit of nude BALB/c mice, the mice were randomly divided into two groups and intraperitoneally administered DMSO or bortezomib at a dose of 0.5 mg/kg every other day for 30 days. The volume of xenografts was measured every five days (tumor volume = (length × width²)/2).

Dosage form 0.5 mg/kg; s.c.

Applications Bortezomib administration was very effective in inhibiting tumor growth in vivo throughout the course of treatment, resulting in decreased tumor size.

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References:

- [1]. Wang Y, Liang Y, Zhang Y, Wu D, Liu H. Bortezomib inhibits bone marrow-derived dendritic cells. International journal of clinical and experimental pathology. 2015;8(5):4857.
- [2]. Bao X, Ren T, Huang Y, Ren C, Yang K, Zhang H, Guo W. Bortezomib induces apoptosis and suppresses cell growth and metastasis by inactivation of Stat3 signaling in chondrosarcoma. International journal of oncology. 2016 Dec 14;50(2):477-86.

Background

Bortezomib (PS-341), as a dipeptide boronic acid proteasome inhibitor with antitumor activity, can potently inhibit 20S proteasome with K_i of 0.6 nM by targeting a threonine residue^[1].

In vitro, Bortezomib can retain NF-kappaB in the cytoplasm and inhibit cell growth with IC_{50} of 22.5 nM, in a dose/time-dependent way^[2]. Bortezomib inhibited growth and induced apoptosis of PEL (primary effusion lymphomas) cell lines with IC_{50} values of 3.4-5.0 nM^[3].

In vitro, 1 μ mol/L-10 nmol/L bortezomib can activate the Akt pathway via increased SRC-3 (Steroid receptor coactivator-3)^[4]. In vitro, 20 nM bortezomib induces apoptotic cell death and promotes G0/G1 phase arrest^[5].

In vivo, BALB/c mice were treated with 1 mg/kg bortezomib intraperitoneally once weekly for 2 weeks increased phosphorylation of JNK (c-Jun N-terminal kinase) and extracellular signal-regulated protein kinase (ERK) in the spinal cord^[6]. In vivo, mice were treated with bortezomib (2 mg/kg, i.v.) on Day1 and Day4 each week for continuous 4 weeks. bortezomib-treated mice showed enhanced mechanical hyperalgesia, decreased tail nerve conduction and sciatic nerve demyelination^[7].

References:

- [1] Adams J, et al. Proteasome inhibitors: a novel class of potent and effective antitumor agents. Cancer Res. 1999 Jun 1;59(11):2615-22.
- [2] Galimberti S, et al. PS-341 (Bortezomib) inhibits proliferation and induces apoptosis of

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megakaryoblastic MO7-e cells. Leuk Res. 2008 Jan;32(1):103-12.

[3] An J, et al. Antitumor effects of bortezomib (PS-341) on primary effusion lymphomas. Leukemia. 2004 Oct;18(10):1699-704.

[4] Ayala G, et al. Bortezomib-mediated inhibition of steroid receptor coactivator-3 degradation leads to activated Akt. Clin Cancer Res. 2008 Nov 15;14(22):7511-8.

[5] Bao X, et al. Bortezomib induces apoptosis and suppresses cell growth and metastasis by inactivation of Stat3 signaling in chondrosarcoma. Int J Oncol. 2017 Feb;50(2):477-486.

[6] Tsubaki M., et al. Trametinib suppresses chemotherapy-induced cold and mechanical allodynia via inhibition of extracellular-regulated protein kinase 1/2 activation. Am. J. Cancer Res. 2018;8:1239-1248.

[7] Wu Z, et al. Lysosomal dysfunction in Schwann cells is involved in bortezomib-induced peripheral neurotoxicity. Arch Toxicol. 2023 May;97(5):1385-1396.

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