MG-132

Cat. No.: HY-13259

CAS No.: 133407-82-6

Molecular Formula: $C_{26}H_{41}N_3O_5$

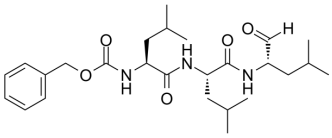
Molecular Weight: 475.62

Target: Proteasome; Autophagy; Apoptosis

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (210.25 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1025 mL	10.5126 mL	21.0252 mL
	5 mM	0.4205 mL	2.1025 mL	4.2050 mL
	10 mM	0.2103 mL	1.0513 mL	2.1025 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: 2.5 mg/mL (5.26 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MG-132 (Z-Leu-Leu-Leu-al) is a potent proteasome and calpain inhibitor with IC_{50}s of 100 nM and 1.2 μM, respectively. MG-132 effectively blocks the proteolytic activity of the 26S proteasome complex. MG-132, a peptide aldehyde, also is an autophagy activator[1][2][3]. MG-132 also induces apoptosis[2].

IC_{50} & Target

IC50: 100 nM (Proteasome), 1.2 μM (Calpain)[1][3]

In Vitro

MG-132 (Z-Leu-Leu-Leu-al) initiates neurite outgrowth in PC12 cells at a low concentration (30 nM) and is a very strong inhibitor of 20S proteasome[3].

MG-132 (10 μM; 1 hour) reverses the effects of TNF- α on I κ B degradation and NF-κ B activation in A549 cells[4].

MG-132 (0.75-5 μM; 24 hours) potently induces p53-dependent apoptosis in KIM-2 cells by 26S proteasome inhibition[5].

MG-132 (10-40 μM; 24 hours) significantly reduces the viability of C6 glioma cells in both time- and concentration-dependent

manners and shows the IC_{50} of 18.5 μM at 24 hours[6].
MG-132 (18.5 μM; 24 hours) induces down-regulation of anti-apoptotic proteins Bcl-2 and XIAP and up-regulates expression of pro-apoptotic protein Bax and caspase-3[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	C6 glioma cells
Concentration:	10, 20, 30, 40 μM
Incubation Time:	24 hours
Result:	Significantly reduced the viability of C6 glioma cells beginning at 6 h in both time- and concentration-dependent manners and showed the IC_{50} of 18.5 μM at 24 hours.

Western Blot Analysis[3]

Cell Line:	A549 cells
Concentration:	10 μM
Incubation Time:	1 hour
Result:	Reversed the effects of TNF-α on IκB degradation and resulted in a reversal of TNF-α-induced NF-κB activation.

In Vivo

MG132 (10 mg/kg; i.p.; daily for 25 days starting 5 days after EC9706 cells injection) significantly inhibits tumor growth of the EC9706 xenograft without causing toxicity to mice[7].
MG-132 (1 mg/kg; i.v.; twice a week for 4 weeks) shows potent tumor inhibitory effect against mice bearing HeLa tumors[8].
MG-132 (1-10 μg/kg/24 hours; subcutaneously implanted osmotic pumps; for 8 days) greatly increases the expression levels of β-dystroglycan, α-dystroglycan, α-sarcoglycan, and dystrophin in skeletal muscle lysates in mice (six-month-old male C57BL/10ScSn DMD mdx mice)[9].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	5- to 6-weeks old female athymic nude mice (EC9706 xenograft)
Dosage:	10 mg/kg
Administration:	I.p.; daily for 25 days starting 5 days after EC9706 cells injection
Result:	Significantly inhibited tumor growth of the EC9706 xenograft without causing toxicity to the mice.

Animal Model:	Five-week-old female C.B-17/Icr-scid/scidJcl mice (bearing HeLa cells)[8]
Dosage:	1 mg/kg
Administration:	Intravenous injection; twice a week for 4 weeks
Result:	The growth inhibition rates in HeLa tumors was 49% compared to the control.

CUSTOMER VALIDATION

- Science. 2020 Dec 4;370(6521):eaay2002.
- Cell Metab. 2021 May 4;33(5):971-987.e6.
- Cell Res. 2021 Sep 3.
- Cell Res. 2021 Mar;31(3):291-311.
- Nat Cell Biol. 2021 Mar;23(3):257-267.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Harhouri K, et al. MG132-induced progerin clearance is mediated by autophagy activation and splicing regulation. EMBO Mol Med. 2017 Sep;9(9):1294-1313.

[2]. Han YH, et al. The effect of MG132, a proteasome inhibitor on HeLa cells in relation to cell growth, reactive oxygen species and GSH. Oncol Rep. 2009 Jul;22(1):215-21.

[3]. Fan WH, et al. Proteasome inhibitor MG-132 induces C6 glioma cell apoptosis via oxidative stress. Acta Pharmacol Sin. 2011 May;32(5):619-25.

[4]. Matsumoto Y, et al. Enhanced efficacy against cervical carcinomas through polymeric micelles physically incorporating theproteasome inhibitor MG132. Cancer Sci. 2016 Jun;107(6):773-81.

[5]. Tsubuki S, et al. Differential inhibition of calpain and proteasome activities by peptidyl aldehydes of di-leucine and tri-leucine. J Biochem. 1996 Mar;119(3):572-6.

[6]. Fiedler MA, et al. Inhibition of TNF-alpha-induced NF-kappaB activation and IL-8 release in A549 cells with the proteasome inhibitor MG-132. Am J Respir Cell Mol Biol. 1998 Aug;19(2):259-68.

[7]. MacLaren AP, et al. p53-dependent apoptosis induced by proteasome inhibition in mammary epithelial cells. Cell Death Differ. 2001 Mar;8(3):210-8.

[8]. Dang L, et al. Proteasome inhibitor MG132 inhibits the proliferation and promotes the cisplatin-inducedapoptosis of human esophageal squamous cell carcinoma cells. Int J Mol Med. 2014 May;33(5):1083-8.

[9]. Bonuccelli G, et al. Proteasome inhibitor (MG-132) treatment of mdx mice rescues the expression and membrane localization of dystrophin and dystrophin-associated proteins. Am J Pathol. 2003 Oct;163(4):1663-75.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA