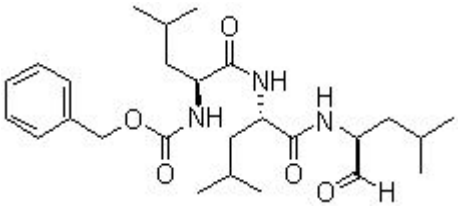
Product Introduction

MG-132

MG-132 is an inhibitor of **proteasome** with **IC50** of 100 nM, and also inhibits calpain with IC50 of 1.2 μM.

Technical Data:

Molecular Weight (MW):	475.62	
Formula:	C26H41N3O5	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 95 mg/mL (199 mM)	
	Water <1 mg/mL (<1 mM)	
	Ethanol 95 mg/mL (199 mM)	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	133407-82-6	

Biological Activity

MG-132 displays >1000 times more activity than ZLLal in inhibiting the ZLLL-MCA-degrading activity of 20S proteasome with IC50 of 100 nM versus 110 μM. MG-132 also inhibits calpain with IC50 of 1.2 μM. MG-132 induces neurite outgrowth in PC12 cells at an optimal concentration of 20 nM, displaying 500

times more potency than ZLLal. [1] MG-132 (10 μM) potently inhibits TNF-α-induced NF-κB activation, interleukin-8 (IL-8) gene transcription, and IL-8 protein release in A549 cells by inhibition of proteasome-mediated IκBα degradation. [2] MG-132 treatment potently induces p53-dependent apoptosis in KIM-2 cells by 26S proteasome inhibition. [3] Unlike BzLLLCOCHO or PS-341, MG-132 treatment results in weak inhibition of the chymotrypsinlike (CT-L) and peptidylglutamyl peptide hydrolysing (PGPH) activities of the 26S proteasome, whereas multiple myeloma cells (U266 and OPM-2) are more sensitive to induction of apoptosis by MG-132 than BzLLLCOCHO. [4] MG-132 (1 μM) sensitizes TRAIL-resistant prostate cancer cells by activating the AP-1 family members c-Fos and c-Jun, which, in turn, repress the antiapoptotic molecule c-FLIP(L). [5] MG-132 significantly enhances the ability of inositol hexakisphosphate (IP6) to reduce cellular metabolic activity in both PC3 and DU145 androgen-independent prostate cancer (AIPCa) cell lines. [6]

Administration of MG-132 effectively rescues the expression levels and plasma membrane localization of dystrophin, β-dystroglycan, α-bdystroglycan, and α-sarcoglycan in skeletal muscle fibers from mdx mice, reduces muscle membrane damage, and ameliorates the histopathological signs of muscular dystrophy. [7] MG-132 treatment significantly reduces immobilization-induced skeletal muscle atrophy in mice, by downregulating the muscle-specific ubiquitin ligases atrogin-1/MAFbx and MuRF-1 mRNA. [8]

References

[1] Tsubuki S, et al. J Biochem, 1996, 119(3), 572-576.

[2] Fiedler MA, et al. Am J Respir Cell Mol Biol, 1998, 19(2), 259-268.

[3] MacLaren AP, et al. Cell Death Differ, 2001, 8(3), 210-218.

[4] Crawford LJ, et al. Cancer Res, 2006, 66(12), 6379-6386.

[5] Li W, et al. Cancer Res, 2007, 67(5), 2247-2255.

[6] Diallo JS, et al. Br J Cancer, 2008, 99(10), 1613-1622.

[7] Bonuccelli G, et al. Am J Pathol, 2003, 163(4), 1663-1675.

[8] Caron AZ, et al. BMC Musculoskelet Disord, 2011, 12:185.

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