

# MG-132

Catalog No: tcsc0471



## Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg



## Specifications

### CAS No:

133407-82-6

### Formula:

$C_{26}H_{41}N_3O_5$

### Pathway:

Metabolic Enzyme/Protease;Autophagy

### Target:

Proteasome;Autophagy

### Purity / Grade:

>98%

### Solubility:

DMSO :  $\geq 160$  mg/mL (336.40 mM)

### Observed Molecular Weight:

475.62

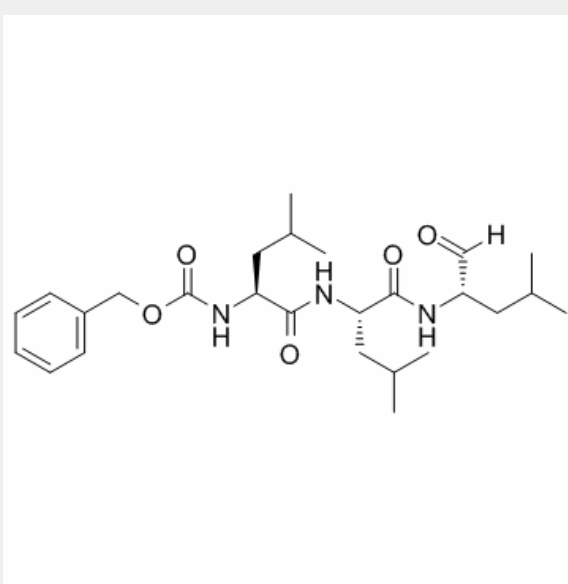
## Product Description

MG-132 is a potent, non-specific **20S proteasome** inhibitor, with **IC<sub>50</sub>** of 24.2 nM for the  $\beta 5$  **chymotrypsin**-like active site.

IC50 & Target: IC50: 24.2 nM (chymotrypsin-like activity)<sup>[1]</sup>

**In Vitro:** Dose-dependent inhibition of cell growth is observed in HeLa cells with an IC<sub>50</sub> of approximately 5 μM MG132 for 24 h. MG132 inhibits the growth of HeLa cells via inducing the cell cycle arrest as well as triggering apoptosis<sup>[2]</sup>. MG-132 inhibits C6 glioma cell proliferation in a time- and dose-dependent manner (the IC<sub>50</sub> value at 24 h is 18.5 μM). MG-132 (18.5 μM) suppresses the proteasome activity by about 70% at 3 h. MG-132 induces apoptosis via down-regulation of antiapoptotic proteins Bcl-2 and XIAP, up-regulation of pro-apoptotic protein Bax and caspase-3, and production of cleaved C-terminal 85 kDa PARP. MG-132 also causes a more than 5-fold increase of reactive oxygen species<sup>[3]</sup>. The IC<sub>50</sub> of MG-132 against HeLa, CaSki, and C33A cervical cancer cells viability after 48 h of incubation is 2.1, 3.2, and 5.2 μM, respectively<sup>[4]</sup>.

**In Vivo:** The in vivo antitumor activity of MG-132 against cervical cancer is examined using s.c. xenograft models. MG-132 is injected at 1 mg/kg using the following schedule: days 1, 4, 8, 12, 15, 18, 23, and 26 for mice bearing HeLa tumors. The growth inhibition rates of MG132 compared to control is 49%<sup>[4]</sup>. MG-132 (i.p., 0.1 mg/kg/day) attenuates pressure-overload-induced cardiac hypertrophy and improves cardiac function in abdominal aortic banding (AAB) rats through regulation of ERK1/2 and JNK1 signaling pathways<sup>[5]</sup>.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!