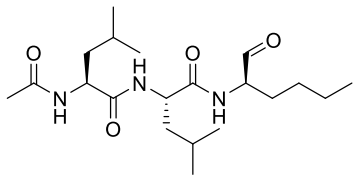
Product data sheet

MedKoo Cat#: 406548 Name: MG-101 CAS#: 110044-82-1 Chemical Formula: $C_{20}H_{37}N_3O_4$ Exact Mass: 383.27841 Molecular Weight: 383.53	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

MG-101, also known as Calpain Inhibitor I and ALLN, is a calpain inhibitor (IC50 = 0.09 μM) that activates p53-dependent apoptosis in tumor cell lines. Activities of MG-101 includes: (1) reduce colon injury caused by dinitrobenzene sulphonic acid; (2) overcome acquired resistance to TRAIL; (3) protect against atractyloside-induced toxicity. (4). reduce colon injury caused by dinitrobenzene sulphonic acid.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	37.56	97.93
DMF	20.0	52.15
Ethanol	48.0	121.15
Ethanol:PBS (pH 7.2) (1:1)	0.5	1.30

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.61 mL	13.04 mL	26.07 mL
5 mM	0.52 mL	2.61 mL	5.21 mL
10 mM	0.26 mL	1.30 mL	2.61 mL
50 mM	0.05 mL	0.26 mL	0.52 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Deissler HL, Lang GK, Lang GE. Fate of the Fc fusion protein aflibercept in retinal endothelial cells: competition of recycling and degradation. Graefes Arch Clin Exp Ophthalmol. 2019 Jan;257(1):83-94. doi: 10.1007/s00417-018-4166-7. Epub 2018 Oct 26. PMID: 30367290; PMCID: PMC6323079.

2. Floyd ZE, Staszkiewicz J, Power RA, Kilroy G, Kirk-Ballard H, Barnes CW, Strickler KL, Rim JS, Harkins LL, Gao R, Kim J, Eilertsen KJ. Prolonged proteasome inhibition cyclically upregulates Oct3/4 and Nanog gene expression, but reduces induced pluripotent stem cell colony formation. Cell Reprogram. 2015 Apr;17(2):95-105. doi: 10.1089/cell.2014.0030. Erratum in: Cell Reprogram. 2015 Aug;17(4):323. Floyd, Elizabeth Z [corrected to Floyd, Z Elizabeth]. PMID: 25826722; PMCID: PMC4378358.

In vivo study

Product data sheet

1. Yoshikawa Y, Hagihara H, Ohga Y, Nakajima-Takenaka C, Murata KY, Taniguchi S, Takaki M. Calpain inhibitor-1 protects the rat heart from ischemia-reperfusion injury: analysis by mechanical work and energetics. Am J Physiol Heart Circ Physiol. 2005 Apr;288(4):H1690-8. doi: 10.1152/ajpheart.00666.2004. Epub 2004 Nov 4. PMID: 15528229.

7. Bioactivity

Biological target:

MG-101 is an inhibitor of cysteine proteases which inhibits calpain I, calpain II, cathepsin B and cathepsin L with Kis of 190, 220, 150 and 500 pM, respectively.

In vitro activity

The cysteine protease inhibitor MG-101 counteracts the activity of lysosomal cathepsins L and B at a concentration of 20 nM or that of the non-lysosomal calpains I and II at 500 nM, respectively. Exposure of iBREC to either effective concentration of MG-101 together with aflibercept for 4 h resulted in a slight but significant increase of aflibercept isolated collectively with proteins from membranes and organelles (Fig. 5a). Because most of the intracellular protein destined to be degraded enters the ubiquitin-proteasome pathway, this study pre-treated iBREC with 20 nM of the inhibitor MG-132 of proteasomal proteases, before aflibercept was added to the cells for 4 h. Western blot analyses of proteins subsequently isolated from the membranes/organelles and the cytoskeleton revealed that more aflibercept accumulated during protease inhibition (Fig. 5b). As was to be expected, the amounts of endogenous proteins actin and claudin-5 were higher in samples isolated from cells treated with the inhibitors MG-101 or MG-132, confirming general inhibition of protein degradation pathways under these conditions (Fig. 5c, d). Higher concentrations of MG-101 or MG-132 could not be used because they resulted in barrier dysfunction and death of iBREC as recognized by cell index measurements.

Reference: Graefes Arch Clin Exp Ophthalmol. 2019; 257(1): 83–94. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6323079/

In vivo activity

CI directly blocked the activation of calpain and prevented the rat heart against the proteolysis of α-fodrin, although CI may not protect the heart from other I/R injury. The present results showing decreased Ca^{2+} handling Vo_2 in E-C coupling with unchanged oxygen costs of PVA and LV contractility probably reflect the decreased total amount of Ca^{2+} handled, which may be due to a suppression of the transsarcolemmal Ca^{2+} influx. The possibility that disruption of cytoskeletal proteins inactivate L-type Ca^{2+} channels has been reported. This study speculates that the linkage of the L-type Ca^{2+} channel to the membrane fodrin acts to tether the channel in place, which somehow modulates the basal activity of the channel, and a loss of the linkage may impair its regulation. Therefore, CI did not induce any conformational changes of the L-type Ca^{2+} channel at the cell membrane, resulting in protection of LV function associated with no impairment of the L-type Ca^{2+} channel function.

Reference: Am J Physiol Heart Circ Physiol. 2005 Apr;288(4):H1690-8. https://pubmed.ncbi.nlm.nih.gov/15528229/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.