Product data sheet



MedKoo Cat#: 510272				
Name: Y27632 HCl				
CAS#: 129830-38-2 (2HCl)				
Chemical Formula: C ₁₄ H ₂₃ Cl ₂ N ₃ O		Ö , N		
Exact Mass: 247.16846		N H-CI		
Molecular Weight: 320.26				
Product supplied as:	Powder)·/// · · · · · · · · · · · · · · · · ·		
Purity (by HPLC):	≥ 98%	i NH ₂		
Shipping conditions	Ambient temperature	14112		
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.			
	In solvent: -80°C 3 months; -20°C 2 weeks.			

1. Product description:

Y27632 is a selective ROCK inhibitor, which inhibits ET-1-induced increases in natriuretic peptide production, cell size, protein synthesis, and myofibrillar organization. Y27632 prevents dimethylnitrosamine-induced hepatic fibrosis in rats, increases apoptosis and disrupts the actin cortical mat in embryonic avian corneal epithelium, affects initial heart myofibrillogenesis in cultured chick blastoderm, promotes the proliferation and cell cycle progression of cultured astrocyte from spinal cord.

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

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Solvent	Max Conc. mg/mL	Max Conc. mM		
DMSO	39.51	123.37		
DMF	20.0	62.45		
Ethanol	8.5	26.54		
PBS (pH 7.2)	10.0	31.22		
Water	73.34	229.0		

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.12 mL	15.61 mL	31.22 mL
5 mM	0.62 mL	3.12 mL	6.24 mL
10 mM	0.31 mL	1.56 mL	3.12 mL
50 mM	0.06 mL	0.31 mL	0.62 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. Li X, Zhou Q, Wang S, Wang P, Li J, Xie Z, Liu C, Wen J, Wu X. Prolonged treatment with Y-27632 promotes the senescence of primary human dermal fibroblasts by increasing the expression of IGFBP-5 and transforming them into a CAF-like phenotype. Aging (Albany NY). 2020 Aug 25;12(16):16621-16646. doi: 10.18632/aging.103910. Epub 2020 Aug 25. PMID: 32843583; PMCID: PMC7485707.

In vivo study

1. Kim K, Min S, Kim D, Kim H, Roh S. A Rho Kinase (ROCK) Inhibitor, Y-27632, Inhibits the Dissociation-Induced Cell Death of Salivary Gland Stem Cells. Molecules. 2021 May 1;26(9):2658. doi: 10.3390/molecules26092658. PMID: 34062818; PMCID: PMC8124333.

7. Bioactivity

Product data sheet



Biological target:

Y-27632 dihydrochloride is an ATP-competitive inhibitor of ROCK-I and ROCK-II, with K_is of 220 and 300 nM, respectively.

In vitro activity

First this study treated HDFs with or without Y-27632 in the presence of 100 ng/ml IGFBP-5, a concentration close to what was found in the conditioned media after Y-27632 treatment. After 48 h, this study analyzed the senescence of HDFs by SA- β -gal staining and found that treatment with Y-27632 and/or IGFBP-5 significantly induced the senescence of HDFs (Figure 7A, 7B). In contrast, when HDFs were treated with Y-27632 with or without the knockdown of IGFBP-5 (siIGFBP-5), which was confirmed by RT-PCR analysis (Supplementary Figure 7A), the knockdown of IGFBP-5 significantly blocked the increased cellular senescence induced by Y-27632 (Figure 7C, 7D). These results suggest that the prolonged treatment of HDFs with Y-27632 increases cellular senescence probably by enhancing the expression and production of IGFBP-5.

Reference: Aging (Albany NY). 2020 Aug 31; 12(16): 16621–16646. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7485707/

In vivo activity

In conclusion, this study demonstrated the role of the ROCK signaling pathway in the survival of mouse SGSCs. By inhibiting the ROCK signaling pathway through the chemical inhibitor Y-27632, dissociation-induced cell death of SGSCs was significantly reduced. In addition, ECM-based experiments revealed that the disruption of the cell–ECM interactions is associated with increased cell death of SGSCs.

Reference: Molecules. 2021 May; 26(9): 2658. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8124333/

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.