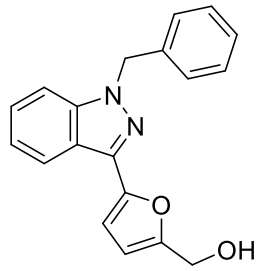
Product data sheet

MedKoo Cat#: 406116 Name: Lificiguat CAS#: 170632-47-0 Chemical Formula: $C_{19}H_{16}N_2O_2$ Exact Mass: 304.12118 Molecular Weight: 304.34	
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:

Lificiguat, also known as YC-1, is a inhibitor of Hypoxia-inducible factor-1alpha (HIF-1alpha). YC-1 is widely used as a potent inhibitor of HIF-1alpha both in vitro and in vivo, and is also being developed as a novel anticancer drug. YC-1 effectively inhibits tumor invasion and metastasis, and imply that YC-1 is worth while to further develop as a multipurpose anticancer drug.

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	53.0	174.15
Ethanol	22.0	72.53

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.29	16.43	32.86
5 mM	0.66	3.29	6.57
10 mM	0.33	1.64	3.29
50 mM	0.07	0.33	0.66

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. Wu JY, Shih YL, Lin SP, Hsieh TY, Lin YW. YC-1 Antagonizes Wnt/β-Catenin Signaling Through the EBP1 p42 Isoform in Hepatocellular Carcinoma. Cancers (Basel). 2019 May 13;11(5):661. doi: 10.3390/cancers11050661. PMID: 31086087; PMCID: PMC6562864.
2. Hsieh KY, Wei CK, Wu CC. YC-1 Prevents Tumor-Associated Tissue Factor Expression and Procoagulant Activity in Hypoxic Conditions by Inhibiting p38/NF-κB Signaling Pathway. Int J Mol Sci. 2019 Jan 9;20(2):244. doi: 10.3390/ijms20020244. PMID: 30634531; PMCID: PMC6359014.

In vivo study

1. Yan Z, An J, Shang Q, Zhou N, Ma J. YC-1 Inhibits VEGF and Inflammatory Mediators Expression on Experimental Central Retinal Vein Occlusion in Rhesus Monkey. Curr Eye Res. 2018 Apr;43(4):526-533. doi: 10.1080/02713683.2018.1426102. Epub 2018 Jan 24. PMID: 29364731.
2. Komsuoglu Celikyurt I, Utkan T, Ozer C, Gacar N, Aricioglu F. Effects of YC-1 on learning and memory functions of aged rats. Med Sci Monit Basic Res. 2014 Aug 21;20:130-7. doi: 10.12659/MSMBR.891064. PMID: 25144469; PMCID: PMC4148360.

Product data sheet

7. Bioactivity

Biological target:

Lificiguat binds to the β subunit of soluble guanylyl cyclase(sGC) with Kd of 0.6-1.1 μM in the presence of CO.

In vitro activity

To further examine the role of YC-1 in the regulation of Wnt signaling, HCC cells were treated with the IC50 of YC-1. The effect of YC-1 on Wnt signaling was evaluated by STF luciferase reporter assays. YC-1 significantly decreased the transcriptional activity of TOPflash but not that of the negative control FOPflash in HepG2, Huh6 and Hep3B cells (Figure 2A). Cyclin D1 is the downstream gene of the Wnt signaling pathway [8,19]. Subsequently, we confirmed that YC-1 decreased the expression of cyclin D1 in HepG2, Huh6 and Hep3B cells in a time-dependent manner (Figure 2B). Considering the above results, this suggests that YC-1 effectively reduces the expression of cyclin D1 through the attenuation of Wnt signaling activation, thereby suppressing tumor cell proliferation.

Cancers (Basel). 2019 May; 11(5): 661. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6562864/

In vivo activity

The aim of this study was to investigate the effects of a potent nitric oxide-guanylate cyclase activator, 3-(5'-hydroxymethyl-2'-furyl)-1-benzylindazole (YC-1), on learning and memory functions in aged rats. Rats were divided into 2 groups as 4-month-old and 24-month-old rats. Rats received YC-1 (1 mg/kg/day) for 2 weeks long-term. There was a significant change in retention latency between the groups (1-way ANOVA, $F(4,45)=45.61$, $p<0.0001$, Figure 4). Post-hoc comparisons showed that retention latency of 24-month-old aged rats was significantly shortened compared to 4-month-old young rats ($p<0.0001$, Dunnett's test) and YC-1-treated 24-month-old rats ($p<0.0001$, Dunnett's test) (Figure 4). The shortened retention latency scores caused by aging is reversed with the administration of YC-1 to aged rats ($p<0.0001$, Dunnett's test). Foot-shock sensitivity results suggest that neither aging nor YC-1 treatment caused gross motor disabilities at testing (Figure 6). The results suggest that improvement of age-related learning and memory deficits via chronic YC-1 treatment may result from the direct effects of YC-1 on sGC.

Med Sci Monit Basic Res. 2014; 20: 130–137. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4148360/