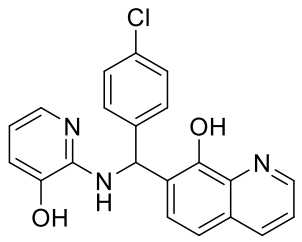
Product data sheet

MedKoo Cat#: 561218 Name: Adaptaquin CAS#: 385786-48-1 Chemical Formula: $C_{21}H_{16}ClN_3O_2$ Exact Mass: 377.0931 Molecular Weight: 377.83	
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:
Adaptaquin is a hydroxyquinoline inhibitor of HIF-PHD enzymes. Adaptaquin reduces neuronal death and behavioral deficits after intracerebral hemorrhage (ICH) in several rodent models without affecting total iron or zinc distribution in the brain.

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	33.89	89.70
DMSO:PBS (pH 7.2) (1:2)	0.33	0.87
DMF	30.0	79.40

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.65 mL	13.23 mL	26.47 mL
5 mM	0.53 mL	2.65 mL	5.29 mL
10 mM	0.26 mL	1.32 mL	2.65 mL
50 mM	0.05 mL	0.26 mL	0.53 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. Aimé P, Karuppagounder SS, Rao A, Chen Y, Burke RE, Ratan RR, Greene LA. The drug adaptaquin blocks ATF4/CHOP-dependent pro-death Trib3 induction and protects in cellular and mouse models of Parkinson's disease. Neurobiol Dis. 2020 Mar;136:104725. doi: 10.1016/j.nbd.2019.104725. Epub 2020 Jan 3. PMID: 31911115; PMCID: PMC7545957.
2. Neitemeier S, Dolga AM, Honrath B, Karuppagounder SS, Alim I, Ratan RR, Culmsee C. Inhibition of HIF-prolyl-4-hydroxylases prevents mitochondrial impairment and cell death in a model of neuronal oxytosis. Cell Death Dis. 2016 May 5;7(5):e2214. doi: 10.1038/cddis.2016.107. PMID: 27148687; PMCID: PMC4917646.

In vivo study

1. Li K, Li T, Wang Y, Xu Y, Zhang S, Culmsee C, Wang X, Zhu C. Sex differences in neonatal mouse brain injury after hypoxia-ischemia and adaptaquin treatment. J Neurochem. 2019 Sep;150(6):759-775. doi: 10.1111/jnc.14790. Epub 2019 Jul 28. PMID: 31188470.
2. Karuppagounder SS, Alim I, Khim SJ, Bourassa MW, Sleiman SF, John R, Thinnes CC, Yeh TL, Demetriades M, Neitemeier S, Cruz D, Gazaryan I, Killilea DW, Morgenstern L, Xi G, Keep RF, Schallert T, Tappero RV, Zhong J, Cho S, Maxfield FR, Holman TR, Culmsee C, Fong GH, Su Y, Ming GL, Song H, Cave JW, Schofield CJ, Colbourne F, Coppola G, Ratan RR. Therapeutic

targeting of oxygen-sensing prolyl hydroxylases abrogates ATF4-dependent neuronal death and improves outcomes after brain hemorrhage in several rodent models. Sci Transl Med. 2016 Mar 2;8(328):328ra29. doi: 10.1126/scitranslmed.aac6008. PMID: 26936506; PMCID: PMC5341138.

7. Bioactivity

Biological target:

HIF-prolyl hydroxylase-2 (PHD2) inhibitor.

In vitro activity

By contrast, cultures treated with 0.5, 1 and 5 μM AQ (Adaptaquin) displayed robust protection from 6-OHDA, both in cell numbers and morphology, with extensive neurite preservation (Fig. 1A,B). This study further confirmed protection by immunostaining phosphorylated (Ser139) histone H2A.X (PH2AX), an apoptotic marker. In cultures treated with 6-OHDA ± AQ (Fig. 1C), 6-OHDA alone induced a robust increase in nuclear PH2AX staining while co-treatment with 0.5 μM AQ blocked this response. Altogether, these results demonstrate that AQ prevents apoptosis of 6-OHDA-treated neuronal PC12 cells.

Reference: Neurobiol Dis. 2020 Mar; 136: 104725. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7545957/

In vivo activity

Adaptaquin-treated mice, however, showed decreased edema 7 days after collagenase injection, likely because adaptaquin, in contrast to the conditional deletion of HIF-PHD isoforms, is available to target vascular and immune cells (fig. S7B). Mice with striatal hemorrhage showed a preference for ipsilateral turns because of deficits in the weight-balancing movements of the limbs contralateral to the injury, as well as spatial neglect. This preference was normalized in adaptaquin-treated mice as measured by the corner turn task (Fig. 4C; $P < 0.01$). Another behavior (tape removal task), which represents a form of sensory neglect, improved significantly in adaptaquin-treated mice 1 and 3 days after ICH (Fig. 4D; $P < 0.05$). Adaptaquin-induced behavioral improvements were associated with a reduction in the number of degenerating neurons in perihematomal and hematomal areas of the mouse striatum (Fig. 4, E to I; $P < 0.001$).

Reference: Sci Transl Med. 2016 Mar 2; 8(328): 328ra29. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5341138/

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.