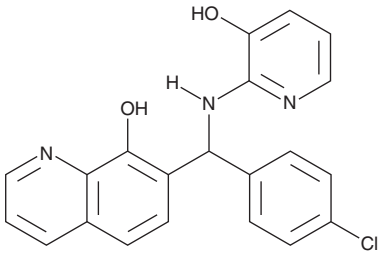
PRODUCT INFORMATION

Adaptaquin

Item No. 19720

CAS Registry No.:	385786-48-1
Formal Name:	7-[(4-chlorophenyl)[(3-hydroxy-2-pyridinyl) amino]methyl]-8-quinolinol
MF:	$C_{21}H_{16}ClN_3O_2$
FW:	377.8
Purity:	≥95%
UV/Vis.:	λ_{max}: 247, 307 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Adaptaquin is supplied as a crystalline solid. A stock solution may be made by dissolving the adaptaquin in the solvent of choice, which should be purged with an inert gas. Adaptaquin is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of adaptaquin in these solvents is approximately 30 mg/ml.

Adaptaquin is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, adaptaquin should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Adaptaquin has a solubility of approximately 0.33 mg/ml in a 1:2 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Adaptaquin is an inhibitor of hypoxia-inducible factor prolyl hydroxylase 2 (HIF-PH2; IC_{50} = 2 micromolar).[1] It stabilizes HIF-1α protein and induces expression of the HIF-1-regulated genes *EPO* and *VEGF* in SH-SY5Y human neuroblastoma cells. Adaptaquin protects against cell death induced by glutathione depletion in rat primary cortical neurons (IC_{50} = 0.25 µM) and reduces glutamate-induced cell death in HT-22 cells.[1,2] *In vivo*, adaptaquin (30 mg/kg) increases pellet retrieval in the single-pellet reaching task in a rat autologous striatal blood infusion model of intracerebral hemmorhage and normalizes preference for ipsilateral turns in a mouse model of striatal hemorrhage.[3]

References

1. Smirnova, N.A., Rakhman, I., Moroz, N., *et al.* Utilization of an in vivo reporter for high throughput identification of branched small molecule regulators of hypoxic adaptation. *Chem. Biol.* **17(4)**, 380-391 (2010).
2. Neitemeier, S., Dolga, A.M., Honrath, B., *et al.* Inhibition of HIF-prolyl-4-hydroxylases prevents mitochondrial impairment and cell death in a model of neuronal oxytosis. *Cell Death Dis.* **7**, e2214 (2016).
3. Karuppagounder, S.S., Alim, I., Khim, S.J., *et al.* 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.* **8(328)**, 328ra2 (2016).

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