Product Data Sheet

Product Name: Adaptaquin
Cat. No.: GC12487

Chemical Properties

Cas. No.	385786-48-1		
Chemical Name	7-[(4-chlorophenyl)[(3-hydroxy-2-pyridinyl)amino]methyl]-8-quinolinol		
SMILES	OC1=CC=CN=C1NC(C2=CC=C(Cl)C=C2)C3=C(O)C(N=CC=C4)=C4C=C3		
Formula	$C_{21}H_{16}ClN_3O_2$	M.Wt	377.8
Solubility	≤30mg/ml in DMSO;30mg/ml in dimethyl formamide	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Adaptaquin		

Background

Adaptaquin is a selective hydroxyquinoline HIF prolyl hydroxylase (HIF-PHD) inhibitor [1] [2].

The hypoxia-inducible factor prolyl hydroxylase domain enzymes (HIF-PHDs) are a family of oxygen sensors that has been implicated in neuronal survival. Catalysis by the HIF-PHDs destabilizes the transcriptional activator HIF-1a under normoxia. HIF-PHDs are promising target candidates for mitochondrial protection in paradigms of oxidative stress. The inhibition of HIF-PHDs prevented neuronal cell death induced by mitochondrial toxins [1][2].

Adaptaquin is a hydroxyquinoline HIF-PHD inhibitor. Adaptaquin inhibited purified and recombinant PHD2. Adaptaquin (30 mg/kg) penetrated the blood-brain barrier, resulting in inhibition of the oxygen-sensing HIF-PHDs and activation of HIF-dependent gene expression [1]. In HT-22 cells, Adaptaquin protected against glutamate-induced cell death. Adaptaquin could also restore the mitochondrial ATP production [2].

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In intracerebral hemorrhage (ICH) mice model, Adaptaquin decreased edema and significantly improved tape removal task, which were associated with a reduction in the number of degenerating neurons in perihematomal and hematomal areas of the mouse striatum [1].

References:

[1]. Karuppagounder SS, Alim I, Khim SJ, 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. 2016 Mar 2;8(328):328ra29.

[2]. Neitemeier S, Dolga AM, 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. 2016 May 5;7:e2214.

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