CC260

Cat. No.:	HY-139188			
CAS No.:	2411088-26-9			
Molecular Formula:	C ₂₄ H ₂₉ Cl ₂ N ₅ O ₂			
Molecular Weight:	490.43			
Target:	PI5P4K			
Pathway:	Metabolic Enzyme/Protease			
Storage:	Powder	-20°C	3 years	
		4°C	2 years	
	In solvent	-80°C	6 months	
		-20°C	1 month	

®

MedChemExpress

SOLVENT & SOLUBILITY

In Vitro	DMSO : 160 mg/mL (3	DMSO : 160 mg/mL (326.24 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg		
		1 mM	2.0390 mL	10.1951 mL	20.3903 mL		
		5 mM	0.4078 mL	2.0390 mL	4.0781 mL		
		10 mM	0.2039 mL	1.0195 mL	2.0390 mL		
	Please refer to the solubility information to select the appropriate solvent.						
In Vivo	 Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 4 mg/mL (8.16 mM); Suspended solution; Need ultrasonic Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 4 mg/mL (8.16 mM); Clear solution 						

Description	CC260 is a selective PI5P4Kα and PI5P4Kβ inhibitor with K _i s of 40 nM and 30 nM, respectively. CC260 does not inhibit or weakly inhibits other protein kinases, such as Plk1 and RSK2. CC260 can be used for cell energy metabolism, diabetes and cancer research ^[1] .			
IC ₅₀ & Target	Ki: 40 nM (PI5P4Ka) and 30 nM (PI5P4K β) ^[1]			
In Vitro	In cultured C2C12 myotubes, CC260 (20 μM) enhances Insulin-induced Akt phosphorylation at both Thr-308 and Ser-473 but suppresses S6K phosphorylation (Thr-389) by mTORC1 ^[1] . CC260 (2.5 μM, 5 μM, 10 μM, 20 μM) significantly increases phosphorylation of acetyl-CoA carboxylase (ACC) in a dose- dependent manner ^[1] .			

CI

CC260 treatment reduces the ability of BT474 cells to survive serum starvation, which could be rescued by expressing the PI5P4K β refractory mutant^[1].

In BT474 cells, CC260 treatment causes an increase in glycolytic ATP production $^{[1]}$.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Song Chen, et al. Pharmacological inhibition of PI5P4Kα/β disrupts cell energy metabolism and selectively kills p53-null tumor cells. Proc Natl Acad Sci U S A. 2021 May 25;118(21):e2002486118.

Caution: Product has not been fully validated for medical applications. For research use only.

 Tel: 609-228-6898
 Fax: 609-228-5909
 E-mail: tech@MedChemExpress.com

 Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA