DCPIB

Cat. No.:	HY-103371		
CAS No.:	82749-70-0		
Molecular Formula:	C ₂₂ H ₂₈ Cl ₂ O ₄		
Molecular Weight:	427		
Target:	Chloride Channel; Potassium Channel		
Pathway:	Membrane Transporter/Ion Channel		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year

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SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (234.19 mM; Need ultrasonic)						
	Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg		
		1 mM	2.3419 mL	11.7096 mL	23.4192 mL		
		5 mM	0.4684 mL	2.3419 mL	4.6838 mL		
		10 mM	0.2342 mL	1.1710 mL	2.3419 mL		
	Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.08 mg/mL (4.87 mM); Clear solution						
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.08 mg/mL (4.87 mM); Clear solution; Need ultrasonic						
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (4.87 mM); Clear solution						

DIOLOGICAL ACTIV						
Description	DCPIB is a selective, reversible and potent inhibitor of volume-regulated anion channels (VRAC). DCPIB voltage-dependently activates potassium channels TREK1 and TRAAK, and inhibits TRESK, TASK1 and TASK3 (IC ₅₀ s: 0.14, 0.95, 50.72 μM, respectively). DCPIB is also a selective blocker of swelling-induced chloride current (I _{Cl,swell}), with an IC ₅₀ of 4.1 μM. DCPIB is a useful tool for investigating structure-function studies of K2P channels ^{[1][2]} .					
IC ₅₀ & Target	IC50⊠0.14 μM (TRESK), 0.95 μM (TASK1), 50.72 μM (TASK3) ^[1] , 4.1 μM (I _{Cl,swell} , CPAE cells) ^[2]					

Product Data Sheet

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In Vitro	DCPIB (10 μM) activates TREK1 and enhances TRAAK currents in COS-7 cells ^[1] . ?DCPIB (10 μM) prominently and reversibly suppresses TRESK currents in COS-7 cells, with an IC ₅₀ of 0.14 μM ^[1] . ?DCPIB (10?μM) displays selectivity for I _{Cl,swell} and has no significant inhibitory effects on I _{Cl,Ca} in CPAE cells ^[2] . ?DCPIB (10?μM, 5 min) has no effect on attenuate subsequent swelling in cardiomyocytes ^[2] . ?DCPIB (10 μM, 3 h) inhibits LPS-induced MAPK activation in BV2 cells ^[3] . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Immunofluorescence ^[3]				
	Cell Line:	BV2 cells			
	Concentration:	10 μΜ			
	Incubation Time:	3 h			
	Result:	Significantly decreased Ki67 positive staining microglia and pro-inflammatory cytokine (TNF- α , IL-1 β) secretion.			
	Western Blot Analysis ^[3]				
	Cell Line:	BV2 cells			
	Concentration:	10 μΜ			
	Incubation Time:	3 h			
	Result:	Inhibited migratory potential of transient OGD (Oxygen-glucose deprivation) exposed BV2 cells.			
In Vivo	LDN-212854 (intracerebroventricular infusion, 1 mM, 10 μL) suppresses microglial activation and ameliorates neuronal damage in rMCAO rats ^[3] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.				
	Animal Model:	Reversible middle cerebral artery occlusion (rMCAO) model ^[3]			
	Dosage:	1 mM, 10 μL			
	Administration:	Administered manually for 20s by intracerebroventricular infusion			
	Result:	Diminished Pyramidal neurons injury induced by rMCAO in the CA1 region.			

CUSTOMER VALIDATION

- Oncol Rep. 2023 Jun;49(6):115.
- Biochem J. 2023 May 2;BCJ20220614.
- Cell Calcium. 2023 Mar 11;111:102715.
- bioRxiv. 2023 Sep 3.

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REFERENCES

[1]. Qingdong Han, et al. DCPIB, a potent volume-regulated anion channel antagonist, attenuates microglia-mediated inflammatory response and neuronal injury following focal cerebral ischemia. Brain Res. 2014 Jan 13;1542:176-85.

[2]. Lv J, et al. DCPIB, an Inhibitor of Volume-Regulated Anion Channels, Distinctly Modulates K2P Channels. ACS Chem Neurosci. 2019 Apr 17.

[3]. Decher N, et al. DCPIB is a novel selective blocker of I(Cl,swell) and prevents swelling-induced shortening of guinea-pig atrial action potential duration. Br J Pharmacol. 2001 Dec;134(7):1467-79.

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

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