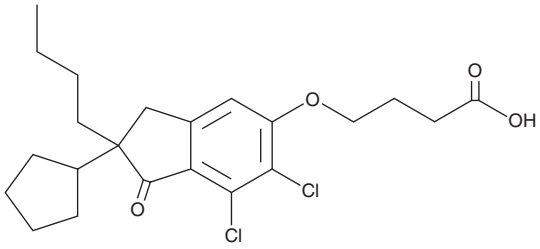
PRODUCT INFORMATION

DCPIB

Item No. 34064

CAS Registry No.:	82749-70-0
Formal Name:	4-[(2-butyl-6,7-dichloro-2-cyclopentyl-2,3-dihydro-1-oxo-1H-inden-5-yl)oxy]-butanoic acid
MF:	$C_{22}H_{28}Cl_2O_4$
FW:	427.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 225, 272 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

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

DCPIB is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, DCPIB should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. DCPIB has a solubility of approximately 0.20 mg/ml in a 1:4 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

DCPIB is an inhibitor of volume-regulated anion channels (VRAC).[1] It inhibits the swelling-induced chloride current ($I_{Cl,swell}$) in bovine pulmonary artery endothelial cells (IC_{50} = 4.1 µM). DCPIB inhibits the VRAC subunit leucine-rich containing 8A (LRRC8A; IC_{50} = 20.9 µM) and inhibits sphingosine-1-phosphate-induced cGAMP uptake by LRRC8A in telomerase-immortalized human microvascular endothelial (TIME) cells when used at a concentration of 20 µM.[2,3] It also inhibits the two-pore domain potassium channels K_{2P}18.1/TRESK, K_{2P}3.1/TASK1, and K_{2P}9.1/TASK3 in COS-7 cells expressing the human channels (IC_{50}s = 0.14, 0.95, and 50.72 µM, respectively).[4] DCPIB (10 µM) activates K_{2P}2.1/TREK1 and K_{2P}4.1/TRAAK channels in COS-7 cells expressing the human channels. Intracisternal administration of DCPIB (20 µg/kg) reduces infarct area and neurological deficit scores in a rat model of cerebral ischemia induced by middle cerebral artery occlusion (MCAO).[5]

References

1. Decher, N., Lang, H.J., Nilius, B., *et al. Br. J. Pharmacol.* **134(7)**, 1467-1479 (2001).
2. Qiu, Z., Dubin, A.E., Mathur, J., *et al. Cell* **157(2)**, 447-458 (2014).
3. Lahey, L.J., Mardjuki, R.E., Wen, X., *et al. Mol. Cell.* **80(4)**, 578-591 (2020).
4. Lv, J., Liang, Y., Zhang, S., *et al. ACS Chem. Neurosci.* **10(6)**, 2786-2793 (2019).
5. Zhang, Y., Zhang, H., Feustel, P.J., *et al. Exp. Neurol.* **210(2)**, 514-520 (2008).

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