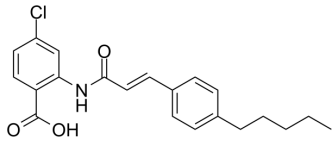
ONO-RS-082

Cat. No.:	HY-123070
CAS No.:	99754-06-0
Molecular Formula:	$C_{21}H_{22}ClNO_3$
Molecular Weight:	371.86
Target:	Phospholipase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (268.92 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6892 mL	13.4459 mL	26.8918 mL
	5 mM	0.5378 mL	2.6892 mL	5.3784 mL
	10 mM	0.2689 mL	1.3446 mL	2.6892 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.72 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (6.72 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

ONO-RS-082 is an inhibitor of phospholipase A (PLA)[1]. ONO-RS-082 inhibits PLA2 with the IC_{50} of 1.0 μM, but does not inhibit PLC even at 100 μM[2].

In Vitro

ONO-RS-082 (10 μM) prevents P. aeruginosa strain PAO1-induced polymorphonumclear cells (PMNs) transepithelial migration, demonstrating that PLA2 activity is crucial to this process[3].

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

Cell Viability Assay[3]

Cell Line:	A549 lung epithelial cell lines

Concentration:	10 μM
Incubation Time:	2-h pretreatment
Result:	Completely blocked HXA_3-mediated PAO1-induced PMN transepithelial migration. Largely prevented PAO1-induced PGE_2 release.

In Vivo

In vivo long-term activation of KCNK3 by ONO-RS-082 (50 mg/kg/day; preventive treatment, day 0 to day 21) reduces the development of PH in the MCT-PH model[4].
In contrast, in vivo short-term KCNK3 activation by ONO-RS-082 (curative treatment) fails to reduce PH symptoms, which is attributed to the complete loss of KCNK3 expression in MCT-PH rats at days 14 to 21[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	MCT- pulmonary hypertension (PH) rat model[4]
Dosage:	50 mg/kg/day
Administration:	
Result:	Reduced the development of PH in the MCT-PH model for long-term.

REFERENCES

[1]. H S Banga,et al. Activation of phospholipases A and C in human platelets exposed to epinephrine: role of glycoproteins IIb/IIIa and dual role of epinephrine. Proc Natl Acad Sci U S A.1986 Dec;83(23):9197-201.

[2]. H Ohno, et al. Effect of phospholipase A2 inhibitors on mouse T lymphocytes. I. Phospholipase A2 inhibitors exert similar immunological activities as glycosylation inhibiting factor. Int Immunol. 1989;1(4):425-33.

[3]. Bryan P Hurley, et al. Selective eicosanoid-generating capacity of cytoplasmic phospholipase A2 in Pseudomonas aeruginosa-infected epithelial cells. Am J Physiol Lung Cell Mol Physiol. 2011 Feb;300(2):L286-94.

[4]. Hélène Le Ribeuz, et al. Implication of Potassium Channels in the Pathophysiology of Pulmonary Arterial Hypertension. Biomolecules. 2020 Sep 1;10(9):1261.

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