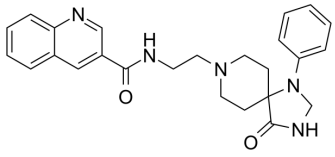
BML-280

Cat. No.:	HY-114095
CAS No.:	1158347-73-9
Molecular Formula:	$C_{25}H_{27}N_5O_2$
Molecular Weight:	429.51
Target:	Phospholipase; TNF Receptor; Interleukin Related
Pathway:	Metabolic Enzyme/Protease; Apoptosis; Immunology/Inflammation
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (291.03 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3282 mL	11.6412 mL	23.2823 mL
	5 mM	0.4656 mL	2.3282 mL	4.6565 mL
	10 mM	0.2328 mL	1.1641 mL	2.3282 mL

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

BIOLOGICAL ACTIVITY

Description

BML-280 (VU0285655-1) is a potent and selective phospholipase D2 (PLD2) inhibitor. BML-280 has the ability to prevent caspase-3 cleavage and reduction in cell viability induced by high glucose. BML-280 can be used for rheumatoid arthritis research[1][2].

IC_{50} & Target	PLD2	PLD1	IL-1β	IL-8

In Vitro

BML-280 shows an approximately 21-fold selectivity for PLD2[3].

BML-280 (0-0.1 μM) suppresses formyl-Met-Leu-Phe (fMLP)-stimulated PLD activity in a concentration dependent manner, with an IC_{50} of 0.04 ± 0.01 μM[3].

BML-280 (0-0.3 μM) inhibits O_2- generation, and the inhibition reaches a plateau (about 20 % inhibition) at around 0.01 μM to 0.3 μM[3].

BML-280 (0-5 μM, 24 h) reduces proliferation in PLD1-deficient cells, but also in PLD2-deficient cells exposed to IGF-1 (Insulin-like growth factor 1)[1].

BML-280 inhibits mRNA levels and secretion of tumor necrosis factor-α, IL-1β and IL-8 in human periodontal ligament cells[2].

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

Cell Proliferation Assay[1]

Cell Line:	Wild-type, PLD1- and PLD2-deficient astrocytes
Concentration:	0, 50, 500, and 5000 nM
Incubation Time:	24 h
Result:	Had minor effects in wild-type and PLD2-deficient cells, but completely blocked PLD activity in PLD1-deficient cells. Caused a highly significant inhibition of glial proliferation when astrocytes were stimulated by FCS (fetal calf serum) or IGF-1, respectively. Showed non-specific effects because they inhibited cell proliferation even in PLD1/2 double knockouts at 5 μM.

REFERENCES

[1]. Burkhardt U, et al. Role of phospholipases D1 and 2 in astroglial proliferation: effects of specific inhibitors and genetic deletion. Eur J Pharmacol. 2015 Aug 15;761:398-404.

[2]. Tenconi PE, et al. High glucose-induced phospholipase D activity in retinal pigment epithelium cells: New insights into the molecular mechanisms of diabetic retinopathy. Exp Eye Res. 2019 Jul;184:243-257.

[3]. Tsai YR, et al. Inhibition of formyl peptide-stimulated phospholipase D activation by Fal-002-2 via blockade of the Arf6, RhoA and protein kinase C signaling pathways in rat neutrophils. Naunyn Schmiedebergs Arch Pharmacol. 2013 Jun;386(6):507-19.

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