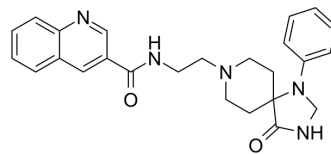


BML-280

Cat. No.:	HY-114095
CAS No.:	1158347-73-9
Molecular Formula:	C ₂₅ H ₂₇ N ₅ O ₂
Molecular Weight:	429.51
Target:	Phospholipase; TNF Receptor; Interleukin Related
Pathway:	Metabolic Enzyme/Protease; Apoptosis; Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



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]} .											
IC₅₀ & Target	PLD2	PLD1	TNF-α	IL-1β								
	IL-8											
In Vitro	<p>BML-280 shows an approximately 21-fold selectivity for PLD2^[3].</p> <p>BML-280 (0-0.1 μM) suppresses formyl-Met-Leu-Phe (fMLP)-stimulated PLD activity in a concentration dependent manner, with an IC₅₀ of 0.04 ± 0.01 μM^[3].</p> <p>BML-280 (0-0.3 μM) inhibits O₂- generation, and the inhibition reaches a plateau (about 20 % inhibition) at around 0.01 μM to 0.3 μM^[3].</p> <p>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].</p> <p>BML-280 inhibits mRNA levels and secretion of tumor necrosis factor-α, IL-1β and IL-8 in human periodontal ligament cells^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Proliferation Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Wild-type, PLD1- and PLD2-deficient astrocytes</td> </tr> <tr> <td>Concentration:</td> <td>0, 50, 500, and 5000 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> <tr> <td>Result:</td> <td>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.</td> </tr> </table>				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.
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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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Caution: Product has not been fully validated for medical applications. For research use only.

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