C16-PAF

Cat. No.: HY-108635

CAS No.: 74389-68-7

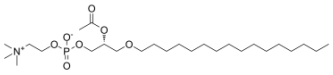
Molecular Formula: $C_{26}H_{54}NO_7P$

Molecular Weight: 523.68

Target: p38 MAPK; MEK; ERK; Endogenous Metabolite

Pathway: MAPK/ERK Pathway; Stem Cell/Wnt; Metabolic Enzyme/Protease

Storage: -20°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (95.48 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9096 mL	9.5478 mL	19.0956 mL
	5 mM	0.3819 mL	1.9096 mL	3.8191 mL
	10 mM	0.1910 mL	0.9548 mL	1.9096 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 (4.77 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.77 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

C16-PAF (PAF (C16)), a phospholipid mediator, is a platelet-activating factor and ligand for PAF G-protein-coupled receptor (PAFR). C16-PAF exhibits anti-apoptotic effect and inhibits caspase-dependent death by activating the PAFR. C16-PAF is a potent MAPK and MEK/ERK activator. C16-PAF induces increased vascular permeability[1][2][3][4][5].

IC_{50} & Target			
	Human Endogenous Metabolite	ERK	MEK

In Vitro

C16-PAF (PAF (C16); 0.5-1.5 μM; for 24 hours) elicits significant concentration-dependent neuronal loss in $PAFR^{-/-}$ but not $PAFR^{+/+}$ cultures. C16-PAF (1 μM) elicits neuronal death in $PAFR^{-/-}$ cells infected with EGFP alone[1].
C16-PAF (1 μM; for 24 hours) activates caspase 7 but not caspase 3 in $PAFR^{-/-}$ neurons[1].

C16-PAF is synthesized by two distinct pathways; the remodeling pathway and the de novo synthesis pathway. C16-PAF acts by binding to a unique G-protein-coupled seven transmembrane receptor[2][3].
C16-PAF (1-25 μg/ml; 6, 12, 24 h) inhibits M. smegmatis and M. bovis BCG growth in a time-dependent manner[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Cerebellar granule neurons (CGNs) from $PAFR^{-/-}$ and $PAFR^{+/+}$ mice
Concentration:	0.5-1.5 μM
Incubation Time:	24 hours
Result:	Elicited significant concentration-dependent neuronal loss in $PAFR^{-/-}$ but not $PAFR^{+/+}$ cultures in serum-free media.

Western Blot Analysis[1]

Cell Line:	CGNs
Concentration:	1 μM
Incubation Time:	24 hours
Result:	Activated caspase 7 but not caspase 3 in $PAFR^{-/-}$ neurons.

REFERENCES

[1]. Scott D Ryan, et al. Heterogeneity in the sn-1 carbon chain of platelet-activating factor glycerophospholipids determines pro- or anti-apoptotic signaling in primary neurons. J Lipid Res. 2008 Oct;49(10):2250-8.

[2]. Z Honda, et al. Transfected platelet-activating factor receptor activates mitogen-activated protein (MAP) kinase and MAP kinase kinase in Chinese hamster ovary cells. J Biol Chem. 1994 Jan 21;269(3):2307-15.

[3]. Muhammad S Riaz, et al. Direct Growth Inhibitory Effect of Platelet Activating Factor C-16 and Its Structural Analogs on Mycobacteria. Front Microbiol. 2018 Sep 11;9:1903.

[4]. Jing Chu, et al. IGHG1 Regulates Prostate Cancer Growth via the MEK/ERK/c-Myc Pathway. Biomed Res Int. 2019 Jul 4;2019:7201562.

[5]. Nina Bögershausen, et al. RAP1-mediated MEK/ERK pathway defects in Kabuki syndrome. J Clin Invest. 2015 Sep;125(9):3585-99.

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