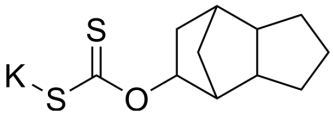
D609

Cat. No.:	HY-70072
CAS No.:	83373-60-8
Molecular Formula:	$C_{11}H_{15}KOS_2$
Molecular Weight:	266.46
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 (375.29 mM; Need ultrasonic)

H_2O : 2 mg/mL (7.51 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7529 mL	18.7645 mL	37.5291 mL
5 mM	0.7506 mL	3.7529 mL	7.5058 mL
10 mM	0.3753 mL	1.8765 mL	3.7529 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 25 mg/mL (93.82 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3 mg/mL (11.26 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (11.26 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (11.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

D609, an antitumoural xanthate, is a specific and competitive phosphatidyl choline-specific phospholipase C (PC-PLC) inhibitor with a K_i of 6.4 μM. D609 is an antioxidative protector and has antiviral and anti-inflammatory activity[1][2][3].

IC_{50} & Target

Ki: 6.4 μM (PC-PLC)

In Vitro

D609 (100 μM; for 2 h) significantly attenuats the proliferation of various cell lines[2].
D609 (50, 100 and 200 μM; for 2 h) results in caspase-3 activation with 200 μM and causes no detectable cleavage with 50, 100 μM[2].
D609 (100 μM; for 2 h) significantly inhibits BrdU incorporation in BV-2 microglia and causes accumulation of cells in G1 phase with decreased number of cells in the S phase[2].
D609 (100 μM; for 2 h and cultured for an additional 2 h or 22 h without D609) increases ceramide levels, up-regulates p21 expression and causes a decreases in phospho-Rb[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	RAW 264.7 macrophages, N9 and BV-2 microglia, and DITNC1 astrocytes
Concentration:	100 μM
Incubation Time:	For 2 hours
Result:	Significantly attenuated the proliferation of RAW 264.7 macrophages, N9 and BV-2 microglia, and DITNC1 astrocytes, without affecting cell viability.

Apoptosis Analysis[2]

Cell Line:	BV-2 cells
Concentration:	50, 100 and 200 μM
Incubation Time:	For 2 hours
Result:	Activated caspase-3 in a dose- and time-dependent manner.

Cell Cycle Analysis[2]

Cell Line:	BV-2 cells
Concentration:	100 μM
Incubation Time:	For 2 hours
Result:	Significantly inhibited BrdU incorporation in BV-2 microglia and caused accumulation of cells in G1 phase with decreased number of cells in the S phase.

Western Blot Analysis[2]

Cell Line:	BV-2 cells
Concentration:	100 μM
Incubation Time:	For 2 hours
Result:	Increased ceramide levels, up-regulated p21 expression and causes a decreased in phospho-Rb.

In Vivo

D609 (2.5, 10 mg/kg/day; ip; for 6 weeks) inhibits the progression of preexisting atherosclerotic lesions in apoE$^{-/-}$ mice and changes the lesion composition into a more stable phenotype[3].
D609 (50 mg/kg; ip; single dose) for 30 min before intratracheal administration of LPS (3 mg/kg) prevents the development of LPS-induced pulmonary hypertension in adult male Wistar rats[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	26-week-old apoE$^{-/-}$ and C57BL/6 WT mice[3]
Dosage:	2.5, 10 mg/kg
Administration:	IP; per day for 6 weeks
Result:	Inhibited the progression of preexisting atherosclerotic lesions in apoE$^{-/-}$ mice and changed the lesion composition into a more stable phenotype. Significantly decreased the aortic endothelial expression of the vascular cell adhesion molecule-1 and the intercellular adhesion molecule-1.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2023 Jul 3;e2206238.
- Traffic. 2015 May;16(5):476-92.
- Bioorg Med Chem. 2015 Sep 15;23(18):6173-84.
- J Physiol Biochem. 2022 Jan 20.
- Thromb J. 2021 Apr 28;19(1):27.

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REFERENCES

[1]. Rachele Pandolfi, et al. Role of acid sphingomyelinase and IL-6 as mediators of endotoxin-induced pulmonary vascular dysfunction. Thorax. 2017 May;72(5):460-471.

[2]. Kalluri HS, et al. D609 inhibits the proliferation of neural progenitor cells.Neuroreport. 2010 Jul 14;21(10):700-3.

[3]. E Amtmann, et al. The antiviral, antitumoural xanthate D609 is a competitive inhibitor of phosphatidylcholine-specific phospholipase C. Drugs Exp Clin Res. 1996;22(6):287-94.

[4]. Lu Zhang, et al. D609 inhibits progression of preexisting atheroma and promotes lesion stability in apolipoprotein e-/- mice: a role of phosphatidylcholine-specific phospholipase in atherosclerosis. Arterioscler Thromb Vasc Biol. 2010 Mar;30(3):411-8.

[5]. Gusain A, et al. Anti-proliferative effects of tricyclodecan-9-yl-xanthogenate (D609) involve ceramide and cell cycle inhibition.Mol Neurobiol. 2012 Jun;45(3):455-64. Epub 2012 Mar 14.

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