Product Data Sheet

Product Name: Z-DQMD-FMK
Cat. No.: GC16744

Chemical Properties

Cas. No.	
Chemical Name	methyl (3S)-3-[[(2S)-2-[[(2S)-5-amino-2-[[(2S)-4-methoxy-4-oxo-2-(phenylmethoxycarbonylamino)butanoyl]amino]-5-oxopentanoyl]amino]-4-methylsulfanylbutanoyl]amino]-5-fluoro-4-oxopentanoate
SMILES	COC(=O)CC(C(=O)CF)NC(=O)C(CCSC)NC(=O)C(CCC(=O)N)NC(=O)C(CC(=O)OC)NC(=O)OCC1=CC=CC=C1
Formula	$C_{29}H_{40}FN_5O_{11}S$
M.Wt	685.72
Solubility	≥ 29.2mg/mL in DMSO
Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.
Structure	Z-DQMD-FMK

Protocol

Cell experiment [1]:

Cell lines	3T3-Swiss Albino cells
Preparation method	The solubility of this compound in DMSO is > 10 mM. General tips for obtaining a higher concentration: Please warm the tube at 37 °C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below - 20 °C for several months.
Reacting condition	25 μM; 24 hrs
Applications	In zinc-deficient 3T3-Swiss Albino cells, Z-DQMD-FMK (25 μM) prevented activation of caspase-3. Z-DQMD-FMK treatment did not restore cell number, but resulted in processing of full-length PKC-δ to a 56-kDa fragment.

References:

[1]. Susan S. CHOU*, Michael S. CLEGG, Alterations in protein kinase C activity and processing during zinc-deficiency-induced cell death, Biochem. J. (2004) 383, 63–71.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

Background

Inhibition of caspase-3 processing by Z-DQMD-FMK (Z-Asp(OMe)-Gln-Met-Asp(OMe)-fluoromethylketone) did not restore cell number in the zinc-deficient group, but resulted in processing of full-length PKC-δ to a 56-kDa fragment[1].

The inhibitory effect of specific caspase inhibitors (Z-DQMD-FMK, Z-IETD-FMK and Z-LEHD-FMK) suggests that the MG132-induced apoptotic cell death and depletion of GSH in SCLC cells are mediated by both activation of caspase-8 and mitochondrial damage, leading to the activation of caspase-9 and -3[2].

To investigate whether εPKC cleavage after stroke is caused by caspase-3 activation, we examined the effect of a cell-permeable caspase-3–specific inhibitor, Z-DQMD-FMK, on generation of cleaved εPKC fragments. Caspase-3 inhibition did not suppress the decrease in fulllength εPKC and the 43-kDa fragment in the ischemic core and penumbra after stroke[3].

References:
1. Susan S. CHOU*, Michael S. CLEGG, Alterations in protein kinase C activity and processing during zinc-deficiency-induced cell death, *Biochem. J*. (2004) 383, 63–71
2. J. H. Banga, E. S. Han. Differential response of MG132 cytotoxicity against small cell lung cancer cells to changes in cellular GSH contents. *Biochemical Pharmacology* 68 (2004) 659–666.
3. T. Shimohata, H. Zhao, εPKC May Contribute to the Protective Effect of Hypothermia in a Rat Focal Cerebral Ischemia Model. *Stroke*. 2007;38:375-380

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA