Product Data Sheet

Product Name: Z-LEHD-FMK TFA
Cat. No.: GC26092

Chemical Properties

Cas. No.	524746-03-0		
Formula	C34H44F4N6O12	M.Wt	804.74
Solubility	Water: 100 mg/mL (124.26 mM);	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-LEHD-FMK TFA		

Protocol

Cell experiment [1]:

Cell lines	HTLV-1-transformed C81 cells (human T-cell leukemia virus type 1-transformed cell line)
Preparation Method	HTLV-1-transformed C81 cells were pretreated with the caspase-9 specific inhibitor Z-LEHD-FMK TFA (100μM) for one hour prior to the addition of the PI3K/AKT inhibitor LY294002.
Reaction Conditions	100μM; 1h pretreatment.
Applications	Pretreatment of the cells with the caspase-9 specific inhibitor Z-LEHD-FMK TFA significantly reduced LY294002-induced apoptosis.

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

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Animal experiment [2]:

Animal models	C57BL/b6N mice
Preparation Method	Mice were pretreated with a single intraperitoneal injection of the caspase-9 inhibitor Z-LEHD-FMK TFA (3mg/kg) 2 hours before the administration of carbon tetrachloride (CCl4) to induce acute liver injury.
Dosage form	3mg/kg; i.p.; single injection.
Applications	Pretreatment with Z-LEHD-FMK TFA significantly exacerbated CCl_4-induced acute hepatocellular injury, as evidenced by increased hepatic necrosis and inflammation, and a marked up-regulation in serum ALT levels. Z-LEHD-FMK TFA also down-regulated the expression of autophagy markers (Beclin1, LC3II, LAMP1), indicating the suppression of cytoprotective autophagy. Z-LEHD-FMK TFA up-regulated the levels of pro-inflammatory mediators (TNF-α, IL-1β) and aggravated hepatic necroptosis via the NF-κB pathway.

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References:
[1] Jeong SJ, Dasgupta A, Jung KJ, et al. PI3K/AKT inhibition induces caspase-dependent apoptosis in HTLV-1-transformed cells. Virology. 2008 Jan 20;370(2):264-72.
[2] Guo R, Lin B, Pan JF, et al. Inhibition of caspase-9 aggravates acute liver injury through suppression of cytoprotective autophagy. Sci Rep. 2016 Sep 1;6:32447.

Background

Z-LEHD-FMK TFA is an irreversible caspase-9 inhibitor. Z-LEHD-FMK TFA prevents apoptosis by inhibiting caspase-9 activity and reduces neural damage by protecting neurons[1-2]. Z-LEHD-FMK TFA can be used in research related to apoptosis and neuroprotection[3-4].

In vitro, pretreatment of HTLV-1-transformed C81 cells with Z-LEHD-FMK TFA (100μM) for 1 hour, followed by stimulation with LY294002 (40μM) for 24-72 hours, significantly prevented LY294002-induced apoptosis[5]. Pretreatment of SGC7901 cells with Z-LEHD-FMK TFA (20μM) for 30 minutes, followed by co-incubation with G503 (20μM) for 24 hours, reduced the rate of G503-induced apoptotic cells[6].

In vivo, a single intraperitoneal injection of Z-LEHD-FMK TFA (3mg/kg) 2 hours before

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CCl_4 injection was administered to C57BL/b6N mice. Z-LEHD-FMK TFA significantly exacerbated CCl_4-induced acute liver injury and down-regulated the expression of hepatoprotective autophagy markers[7]. A single intravenous injection of Z-LEHD-FMK TFA (0.8μmol/kg) administered 1 minute after trauma, or continuous injection for 7 days, was used in rats that suffered thoracic spinal cord contusion. Z-LEHD-FMK TFA effectively blocked post-traumatic apoptosis and was associated with better neurological functional recovery outcomes[8].

References:

[1] Mullani N, Singh MK, Sharma A, et al. Caspase-9 inhibitor Z-LEHD-FMK TFA enhances the yield of in vitro produced buffalo (Bubalus bubalis) pre-implantation embryos and alters cellular stress response. Res Vet Sci. 2016 Feb;104:4-9.
[2] Ozoren N, Kim K, Burns TF, et al. The caspase 9 inhibitor Z-LEHD-FMK TFA protects human liver cells while permitting death of cancer cells exposed to tumor necrosis factor-related apoptosis-inducing ligand. Cancer Res. 2000 Nov 15;60(22):6259-65.
[3] Zhang J, Chen Z, Wang S, et al. Prodelphinidin from purple sweet potato induces apoptosis in human triple-negative breast cancer cells via ROS-mediated ER stress activation. Med Oncol. 2025 Mar 6;42(4):92.
[4] Kim TI, Pak JH, Tchah H, et al. Ceramide-induced apoptosis in rabbit corneal fibroblasts. Cornea. 2005 Jan;24(1):72-9.
[5] Jeong SJ, Dasgupta A, Jung KJ, et al. PI3K/AKT inhibition induces caspase-dependent apoptosis in HTLV-1-transformed cells. Virology. 2008 Jan 20;370(2):264-72.
[6] Huang L, Zhang T, Li S, et al. Anthraquinone G503 induces apoptosis in gastric cancer cells through the mitochondrial pathway. PLoS One. 2014 Sep 30;9(9):e108286.
[7] Guo R, Lin B, Pan JF, et al. Inhibition of caspase-9 aggravates acute liver injury through suppression of cytoprotective autophagy. Sci Rep. 2016 Sep 1;6:32447.
[8] Colak A, Karaoğlan A, Barut S, et al. Neuroprotection and functional recovery after application of the caspase-9 inhibitor Z-LEHD-FMK TFA in a rat model of traumatic spinal cord injury. J Neurosurg Spine. 2005 Mar;2(3):327-34.

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