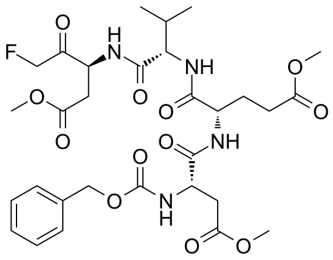
Z-DEVD-FMK

Cat. No.:	HY-12466		
CAS No.:	210344-95-9		
Molecular Formula:	$C_{30}H_{41}FN_4O_{12}$		
Molecular Weight:	668.66		
Target:	Caspase		
Pathway:	Apoptosis		
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 : ≥ 50 mg/mL (74.78 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.4955 mL	7.4776 mL	14.9553 mL
	5 mM	0.2991 mL	1.4955 mL	2.9911 mL
	10 mM	0.1496 mL	0.7478 mL	1.4955 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (1.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (1.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Z-DEVD-FMK is a specific and irreversible caspase-3 inhibitor with an IC_{50} of 18 μM[1].

IC_{50} & Target

Caspase-3
18 μM (IC_{50})

In Vitro

N27 cells are exposed to MPP^+ in the absence or presence of 50 μM Z-DIPD-FMK or 100 μM Z-DEVD-FMK or 50 μM Z-LEHD-FMK and then caspase-9 and caspase-3 enzymatic activities are determined by enzymatic assay at 12 and 24 h following exposure, respectively. Exposure to 300 μM MPP^+ for 24 h in N27 cells results in an approximately 2.5-fold increase in caspase-3 enzyme activity. MPP^+-induced increases in caspase-3 enzyme activity are significantly blocked by 50 μM Z-DIPD-

	FMK, 100 μM Z-DEVD-FMK, and 50 μM Z-LEHD-FMK[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Early Z-DEVD-FMK (160 ng) treatment improves motor and cognitive function after traumatic CNS injury induced by severe controlled cortical impact (CCI) in the mouse[2]. Treatment with Z-DEVD-FMK (160 ng) significantly improves neurological outcome when compared with traumatized animals treated with DMSO vehicle ($p<0.01$)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	N27 cells and primary mesencephalic neurons are exposed to either 10-100 μM 6-OHDA or 10-300 μM MPP^+ in the presence or absence of 0.1-50 μM Z-DIPD-FMK or 0.1-100 μM Z-DEVD-FMK or 50 μM Z-IETD-FMK or Z-LEHD-FMK for the duration of the experiment. N27 cells are incubated with 100 μM 6-OHDA for 24 h or 300 μM MPP^+ for 36 h in the presence or absence of 50 μM Z-DEVD-FMK and cell death is determined by MTT assay, which is widely used to assess cell viability. After treatment, the cells are incubated in serum-free medium containing 0.25 mg/mL MTT for 3 h at 37°C. Formation of formazan from tetrazolium is measured at 570 nm with a reference wavelength at 630 nm using a SpectraMax microplate reader[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2][3]	Mice[2] Male C57Bl/6 mice (20-25 g) are used. For treatment with Z-DEVD-fmk or vehicle after CCI, mice are placed in a stereotaxic apparatus, and the CCI wound is reopened for intracerebroventricular injection. Either Z-DEVD-FMK (160 ng in 2 μL DMSO), or DMSO vehicle is injected over a 5-minute period. Rats[3] Male Sprague Dawley rats (425±25 g) are used. DMSO (5 μL) vehicle or Z-DEVD-FMK (160 ng in 5 μL of DMSO) is administered at a controlled rate of 0.5 μL/min via an infusion pump at 30 min before and at 6 and 24 hr after TBI. At the designated time periods after injury, animals are decapitated under NSC 10816 anesthesia (100 mg/kg, i.p.), and the brains are removed rapidly and dissected. Sham-operated (control) animals received anesthesia and surgery but are not subjected to trauma. Tissue samples are collected 1, 4, 12, 24, and 72 hr after TBI. Samples are frozen on dry ice and kept at −85°C. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Mol Immunol. 2021 Jan;18(1):219-229.
- Nat Commun. 2021 Nov 22;12(1):6786.
- Neuro Oncol. 2023 Apr 21;noad079.
- Acta Pharm Sin B. 2020 Aug;10(8):1397-1413.
- Proc Natl Acad Sci U S A. 2023 May 23;120(21):e2303698120.

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REFERENCES

[1]. Kanthasamy AG, et al. A novel peptide inhibitor targeted to caspase-3 cleavage site of a proapoptotic kinase protein kinase C delta (PKCdelta) protects against dopaminergic neuronal degeneration in Parkinson's disease models. Free Radic Biol Med. 2006 Nov

[2]. Knoblach SM, et al. Caspase inhibitor z-DEVD-fmk attenuates calpain and necrotic cell death in vitro and after traumatic brain injury. J Cereb Blood Flow Metab. 2004 Oct;24(10):1119-32.

[3]. Yakovlev AG, et al. Activation of CPP32-like caspases contributes to neuronal apoptosis and neurological dysfunction after traumatic brain injury. J Neurosci. 1997, 17(19), 7415-7424.

[4]. Huang MY, et al. Chemotherapeutic agent CPT-11 eliminates peritoneal resident macrophages by inducing apoptosis. Apoptosis. 2016 Feb;21(2):130-42.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA