产品名称：Z-DEVD-FMK
产品别名：Z-DEVD-FMK

生物活性：

Description	Z-DEVD-FMK is a specific and irreversible caspase-3 inhibitor with IC50 of 18 μM.
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].
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].

Solvent&Solubility

In Vitro:

DMSO : ≥ 33.33 mg/mL (49.85 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / 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

*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。

储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。

In Vivo:

请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 **In Vitro** 方式配制澄清的储备液，再依次添加助溶剂：

——为保证实验结果的可靠性，澄清的储备液可以根据储存条件，适当保存；体内实验的工作液，建议您现用现配，当天使用； 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比；如在配制过程中出现沉淀、析出现象，可以通过加热和/或超声的方式助溶

1.请依序添加每种溶剂： 10% DMSO→40% PEG300 →5% Tween-80 → 45% saline

Solubility: ≥ 2.5 mg/mL (3.74 mM); Clear solution

此方案可获得 ≥ 2.5 mg/mL (3.74 mM，饱和度未知) 的澄清溶液。

以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 400 μL PEG300 中，混合均匀；向上述体系中加入 50 μL Tween-80，混合均匀；然后继续加入 450 μL 生理盐水定容至 1 mL。

2.请依序添加每种溶剂： 10% DMSO→ 90% (20% SBE-β-CD in saline)

Solubility: ≥ 2.5 mg/mL (3.74 mM); Clear solution

	此方案可获得 ≥ 2.5 mg/mL (3.74 mM，饱和度未知) 的澄清溶液。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 20% 的 SBE-β-CD 生理盐水水溶液中，混合均匀。 3.请依序添加每种溶剂： 10% DMSO →90% corn oil Solubility: ≥ 2.5 mg/mL (3.74 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (3.74 mM，饱和度未知) 的澄清溶液，此方案不适用于实验周期在半个月以上的实验。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中，混合均匀。
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 15;41(10):1578-89. [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.

实验参考：

Cell Assay	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].
Animal Administration	Mice[2] Male C57Bl/6 mice (20-25 g) are used. For treatment with Z-DEVD-fmk or vehicle after CCI, mice are reanesthetized with isoflurane at various times after injury, 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 sodium pentobarbital 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.
	[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

References	degeneration in Parkinson's disease models. Free Radic Biol Med. 2006 Nov 15;41(10):1578-89. [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.