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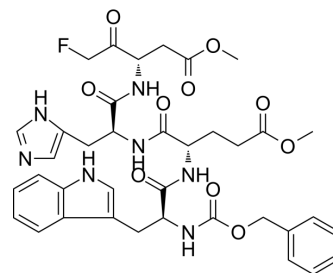
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Z-WEHD-FMK

Cat. No.: HY-P0111
CAS No.: 210345-00-9
Molecular Formula: C₃₇H₄₂FN₇O₁₀
Molecular Weight: 764
Target: Caspase; Cathepsin
Pathway: Apoptosis; Metabolic Enzyme/Protease
Storage: Sealed storage, away from moisture

Powder -80°C 2 years
 -20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (130.89 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		1.3089 mL	6.5445 mL	13.0890 mL
		5 mM		0.2618 mL	1.3089 mL	2.6178 mL
		10 mM		0.1309 mL	0.6545 mL	1.3089 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: ≥ 2.5 mg/mL (3.27 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (3.27 mM); Clear solution					
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (3.27 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Z-WEHD-FMK is a potent, cell-permeable and irreversible caspase-1/5 inhibitor. Z-WEHD-FMK also exhibits a robust inhibitory effect on cathepsin B activity (IC ₅₀ =6 μM). Z-WEHD-FMK can be used to investigate cells for evidence of apoptosis [1][2][4].	
IC ₅₀ & Target	Cathepsin B	Caspase-1
In Vitro	Z-WEHD-FMK (80 μM; 9 hours) elicits a near-complete blockage of C. trachomatis-induced cleavage of golgin-84 and	

increases GM130 expression in cells^[1].

Z-WEHD-FMK (30 min before being exposed to *E. piscicida*) effectively inhibits 0909I *E. piscicida* induced ZF4 cells cytotoxicity and pyroptotic morphology. And in addition, it also inhibits the cytotoxicity induced by cytosolic LPS delivery^[2]. Z-WEHD-FMK (20 μ M; 18-24 hours following Cr^{3+} , Ni^{2+} , and Co^{2+}) significantly induces a decrease of 76% to 86% in IL-1 β release with 200 to 400 ppm Cr^{3+} , it also induces a decrease of 35% to 45% with 48 ppm Ni^{2+} or higher, Finally, this caspase-1 inhibitor induced a decrease with 6 ppm Co^{2+} , down to a level below the detection threshold, and a decrease of 40% to 48% with 12 to 24 ppm Co^{2+} in bone marrow-derived macrophages (BMDM)^[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis^[1]

Cell Line:	C. trachomatis- or mock-infected HeLa cells
Concentration:	80 μ M
Incubation Time:	9 hours
Result:	Increased golgin-84 and GM130 expression.

Cell Viability Assay^[2]

Cell Line:	Mycoplasma free-ZF4 cells
Concentration:	
Incubation Time:	30 min before being exposed to <i>E. piscicida</i>
Result:	Inhibited ZF4 cells cytotoxicity and pyroptotic morphology.

CUSTOMER VALIDATION

- Atherosclerosis. 2019 Oct;289:132-142.

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REFERENCES

- [1]. Kamada S, et al. Caspase-4 and caspase-5, members of the ICE/CED-3 family of cysteine proteases, are CrmA-inhibitable proteases. *Cell Death Differ*. 1997 Aug;4(6):473-8.
- [2]. Yang D, et al. Sensing of cytosolic LPS through casp2 pyrin domain mediates noncanonical inflammasome activation in zebrafish. *Nat Commun*. 2018 Aug 3;9(1):3052.
- [3]. Ferko MA, et al. Effects of metal ions on caspase-1 activation and interleukin-1 β release in murine bone marrow-derived macrophages. *PLoS One*. 2018 Aug 23;13(8):e0199936.
- [4]. Newman ZL, et al. CA-074Me protection against anthrax lethal toxin. *Infect Immun*. 2009 Oct;77(10):4327-36.

Caution: Product has not been fully validated for medical applications. For research use only.

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