

Product Data Sheet

Z-Asp-CH2-DCB

Cat. No.:HY-113953CAS No.:153088-73-4Molecular Formula: $C_{20}H_{17}Cl_2NO_7$ Molecular Weight:454.26

Target: Caspase; Apoptosis

Pathway: Apoptosis

Storage: -20°C, sealed storage, away from moisture

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

SOLVENT & SOLUBILITY

In Vitro

DMSO: 100 mg/mL (220.14 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg
	1 mM	2.2014 mL	11.0069 mL	22.0138 mL
	5 mM	0.4403 mL	2.2014 mL	4.4028 mL
	10 mM	0.2201 mL	1.1007 mL	2.2014 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: ≥ 2.08 mg/mL (4.58 mM); Clear solution
- 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE- β -CD in saline) Solubility: \geq 2.08 mg/mL (4.58 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (4.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Z-Asp-CH2-DCB is an irreversible broad spectrum caspase inhibitor. Z-Asp-CH2-DCB also inhibits proteases with caspase-like activity. Z-D-CH2-DCB blocks the production of IL-1 β , TNF- α , IL-6, and IFN- γ in staphylococcal enterotoxin B (SEB)-stimulated peripheral blood mononuclear cells (PBMC), and reduces SEB-1-stimulated T-cell proliferation in a dose-dependent manner. Z-Asp-CH2-DCB prevents SU5416-induced septal cell apoptosis and emphysema development^{[1][2][3]}.

In Vitro

Z-Asp-CH2-DCB (10-100 μ M) blocks the production of IL-1 β , TNF- α , IL-6, and IFN- γ in SEB-stimulated (200 ng; 16 hours) PBMC in a dose-dependent manner. The production of the chemokines MCP-1, MIP-1 α , and MIP-1 β was also suppresses. The inhibitory effect of Z-Asp-CH2-DCB on TSST-1-activated PBMC is similar, reducing IL-1 β , IL-6, TNF- α , IFN- γ , MCP-1, MIP-1 α , and MIP-1 β to 10, 36, 25, 10, 11, 25, and 30%, respectively, of levels in untreated cells^[1].

	M; 48 hours) inhibits T-cell proliferation in PBMC stimulated with 200 ng of SEB/ml ^[1] . tly confirmed the accuracy of these methods. They are for reference only.	
Cell Viability Assay ^[1]		
Cell Line:	Human peripheral blood mononuclear cells	
Concentration:	10, 50, 100 μΜ	
Incubation Time:	48 hours	
Result:	Inhibited T-cell proliferation in PBMC stimulated with SEB.	

In Vivo

Z-Asp-CH2-DCB (1 mg; i.p.; every day for 3 weeks) prevents SU5416-induced septal cell apoptosis^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (SU5416+ Z-Asp-CH2-DCB group) ^[1]	
Dosage:	1 mg	
Administration:	Intraperitoneal injection; every day for 3 weeks	
Result:	The caspase 3-like activity in SU5416-treated rat lungs is significantly higher, whereas lungs from rats treated with SU5416+Z-Asp-CH2-DCB showed no increase in apoptotic activity.	

REFERENCES

- [1]. Krakauer T, et al. Caspase inhibitors attenuate superantigen-induced inflammatory cytokines, chemokines, and T-cell proliferation. Clin Diagn Lab Immunol. 2004 May;11(3):621-4.
- [2]. Kasahara Y, et al. Inhibition of VEGF receptors causes lung cell apoptosis and emphysema. J Clin Invest. 2000 Dec;106(11):1311-9.
- [3]. Twumasi P, et al. Caspase inhibitors affect the kinetics and dimensions of tracheary elements in xylogenic Zinnia (Zinnia elegans) cell cultures. BMC Plant Biol. 2010 Aug 6;10:162.

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

Tel: 609-228-6898

Fax: 609-228-5909

 $\hbox{E-mail: } tech@MedChemExpress.com$

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