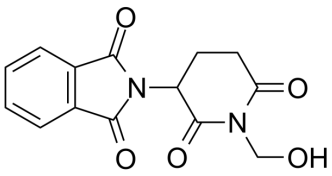
CPS-11

Cat. No.:	HY-117987
CAS No.:	145945-21-7
Molecular Formula:	$C_{14}H_{12}N_2O_5$
Molecular Weight:	288.26
Target:	Nuclear Factor of activated T Cells (NFAT); NF-κB; Reactive Oxygen Species
Pathway:	Immunology/Inflammation; NF-κB; Metabolic Enzyme/Protease
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 57.7 mg/mL (200.17 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4691 mL	17.3455 mL	34.6909 mL
	5 mM	0.6938 mL	3.4691 mL	6.9382 mL
	10 mM	0.3469 mL	1.7345 mL	3.4691 mL

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

BIOLOGICAL ACTIVITY

Description

CPS-11 (N-(Hydroxymethyl)thalidomide) a Thalidomide (HY-14658) analogue, is a potent anti-cancer agent. CPS-11 inhibits NF-κB, activates NFAT, and repress cytokine expression through elevated ROS. CPS-11 exhibits a wider activity spectrum and higher potency against MM (multiple myeloma) cell lines[1][2][3].

In Vitro

CPS-11 abrogates ability of bone marrow stromal cells (BMSCs) to induce proliferation of MM cells, confirming its ability to target tumor cells in the bone marrow microenvironment[1].

CPS-11 (0-200 μM, 0 or 4 h) shows virtually no effect on activation of p38 and only slight, transient activation of ATF2 and HSP27[2].

CPS-11 (0-100 μM, 24 or 48 h) was no toxic to H157 cells, WT MEFs or the p38α-/- MEFs at doses as high as 100 μM[2].

CPS-11 (50 μM, 24 or 48 h) does not induce apoptosis in H157 cells[2].

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

Western Blot Analysis[2]

Cell Line:	H157 cells, PC3 cells, HUVEC cells
Concentration:	0, 1, 5, 10, 20, 50, 100 and 200 μM

Incubation Time:	0, 15 min, 30 min, 1 h, 2 h, and 4 h
Result:	Showed virtually no effect on activation of p38 and only slight, transient activation of ATF2 and HSP27. Increased Akt phosphorylation within 15 minutes, and decreased Akt phosphorylation from 15 minutes to 4 hours.

Cell Cytotoxicity Assay[2]

Cell Line:	H157 cells, wild-type (WT) mouse embryonic fibroblasts (MEF) and p38α-/- MEF
Concentration:	0, 20, 50, and 100 μM
Incubation Time:	24 or 48 hours
Result:	Showed no toxic to H157 cells at doses as high as 100 μM and an incubation time of 48 hours. had no toxic effect on either the WT MEFs or the p38α-/- MEFs at doses as high as 100 μM.

Apoptosis Analysis[2]

Cell Line:	H157 cells
Concentration:	50 μM
Incubation Time:	24 or 48 hours
Result:	Did not induce apoptosis in H157 cells at either time point.

REFERENCES

[1]. Aragon-Ching JB, et al. Thalidomide analogues as anticancer drugs. Recent Pat Anticancer Drug Discov. 2007 Jun;2(2):167-74.

[2]. Warfel NA, et al. Importance of the stress kinase p38alpha in mediating the direct cytotoxic effects of the thalidomide analogue, CPS49, in cancer cells and endothelial cells. Clin Cancer Res. 2006 Jun 1;12(11 Pt 1):3502-9.

[3]. Ge Y, et al. Selective leukemic-cell killing by a novel functional class of thalidomide analogs. Blood. 2006 Dec 15;108(13):4126-35.

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

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