(S)-Thalidomide

Cat. No.: HY-14658A

CAS No.: 841-67-8

Molecular Formula: $C_{13}H_{10}N_2O_4$

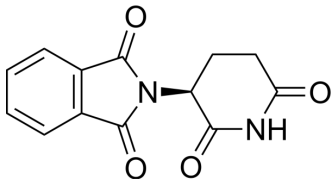
Molecular Weight: 258

Target: Apoptosis; Molecular Glues

Pathway: Apoptosis; PROTAC

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 83.33 mg/mL (322.98 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8760 mL	19.3798 mL	38.7597 mL
	5 mM	0.7752 mL	3.8760 mL	7.7519 mL
	10 mM	0.3876 mL	1.9380 mL	3.8760 mL

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

BIOLOGICAL ACTIVITY

Description

(S)-Thalidomide ((S)-(-)-Thalidomide) is the S-enantiomer of Thalidomide. (S)-Thalidomide has immunomodulatory, anti-inflammatory, antiangiogenic and pro-apoptotic effects[1][2][3]. (S)-Thalidomide induces teratogenic effects by binding to cereblon (CRBN) [4].

IC_{50} & Target

Apoptosis[1]

In Vitro

(S)-Thalidomide treatment results in a reduction in cell viability in U266 cells with an IC_{50} of 362 μM[1].
(S)-Thalidomide treatment increased apoptosis in a dose-dependent manner in U266 cells[1].
There are changes in the expression profile of genes involved in angiogenesis and apoptosis, but the changes are most dramatic in the apoptotic genes. In particular, the expression of I-κB kinase is decreased by two-fold, which is associated with a four-fold decrease in NF-κB expression. (S)-Thalidomide increases the Bax:Bcl-2 ratio, also increases I-kB protein levels, and decreases NF-kB activity. A dramatic decrease in Bcl-2 expression with (S)-Thalidomide suggests a possible enhancement of cytotoxic effect if combined with other cytotoxic agents[1].

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

Cell Viability Assay[1]

Cell Line:	U266 MM cells
Concentration:	0 μM, 10 μM, 100 μM, 150 μM, 200 μM, 1000 μM
Incubation Time:	3 days
Result:	A reduction in cell viability was observed in U266 cells.

Apoptosis Analysis[1]

Cell Line:	U266 MM cells
Concentration:	100 μM, 150 μM, 200 μM, 1000 μM
Incubation Time:	3 days
Result:	Increased apoptosis in U266 cells.

In Vivo

Thalidomide does cause limb reduction defects in chick embryos as long as the embryos are directly exposed to the drug. The most useful techniques are implanting Thalidomide-soaked beads into the embryo immediately adjacent to the limb territory or soaking presumptive chick limb territories in Thalidomide and then grafting the explants to a host embryo celom. Thalidomide affects the chick limb grafted to a host embryo in a dose response fashion. Furthermore, (S)-Thalidomide is more teratogenic than (R)-Thalidomide[1].

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

REFERENCES

[1]. Stephens TD. The effect of thalidomide in chicken embryos. Birth Defects Res A Clin Mol Teratol. 2009 Aug;85(8):725-31.

[2]. Murphy S, et al. Enantioselectivity of thalidomide serum and tissue concentrations in a rat glioma model and effects of combination treatment with cisplatin and BCNU. J Pharm Pharmacol. 2007 Jan;59(1):105-14.

[3]. Liu WM, et al. s-thalidomide has a greater effect on apoptosis than angiogenesis in a multiple myeloma cell line. Hematol J. 2004;5(3):247-54.

[4]. Tokunaga E, et al. Understanding the Thalidomide Chirality in Biological Processes by the Self-disproportionation of Enantiomers. Sci Rep. 2018 Nov 20;8(1):17131.

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