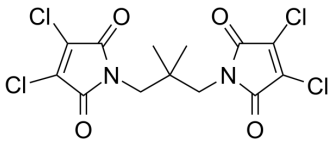
NSC 617145

Cat. No.:	HY-110185
CAS No.:	203115-63-3
Molecular Formula:	$C_{13}H_{10}Cl_4N_2O_4$
Molecular Weight:	400.04
Target:	DNA/RNA Synthesis
Pathway:	Cell Cycle/DNA Damage
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 1 year -20°C 6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10 mg/mL (25.00 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4998 mL	12.4987 mL	24.9975 mL
	5 mM	0.4999 mL	2.4997 mL	4.9995 mL
	10 mM	0.2500 mL	1.2499 mL	2.4997 mL

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

In Vivo

1. Add each solvent one by one: corn oil
   Solubility: 25 mg/mL (62.49 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.67 mg/mL (4.17 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NSC 617145 is a selective werner syndrome helicase (WRN) helicase inhibitor with an IC_{50} value of 230 nM. NSC 617145 inhibits WRN ATPase, and induces double-strand breaks (DSB) and chromosomal abnormalities. NSC 617145 shows selective for WRN over BLM, FANCJ, ChlR1, RecQ, and UvrD helicases[1].

In Vitro

NSC 617145 (0.75-3 μM; 24-72 hours) shows maximal inhibition of proliferation (98%) at the lowest concentration in a WRN-specific manner in HeLa cells[1].
NSC 617145 (0.75 μM; 6 hours) induces WRN binding to chromatin and proteasomal degradation[1].
In FA-D2$^{-/-}$ cells, NSC 617145 (0.125 μM) acts synergistically with very low concentrations of Mitomycin C to inhibit proliferation in a WRN-dependent manner and induce double-strand breaks (DSB) and chromosomal abnormalities. NSC 617145 exposure results in enhanced accumulation of DNA-PKcs pS2056 foci and Rad51 foci in Mitomycin C-treated FA-

deficient cells, suggesting that WRN helicase inhibition prevents processing of Rad51-mediated recombination products and activates NHEJ[1].

NSC 617145, induces cell cycle arrest and apoptosis in human T-cell leukemia virus type 1 (HTLV-1)-transformed adult T-cell leukemia cells[2].

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

Cell Viability Assay[1]

Cell Line:	HeLa cells
Concentration:	0.75 μM, 1 μM, 1.5 μM, 2 μM, 3 μM
Incubation Time:	24 hours, 48 hours, 72 hours
Result:	Inhibited cell proliferation in a WRN-specific manner.

Western Blot Analysis[1]

Cell Line:	HeLa cells
Concentration:	0.75 μM
Incubation Time:	6 hours
Result:	Caused WRN to become degraded by a proteasome-mediated pathway.

REFERENCES

[1]. Monika Aggarwal, et al. Werner syndrome helicase has a critical role in DNA damage responses in the absence of a functional fanconi anemia pathway. Cancer Res. 2013 Sep 1;73(17):5497-507.

[2]. R Moles, et al. WRN-targeted therapy using inhibitors NSC 19630 and NSC 617145 induce apoptosis in HTLV-1-transformed adult T-cell leukemia cells. J Hematol Oncol. 2016 Nov 9;9(1):121.

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