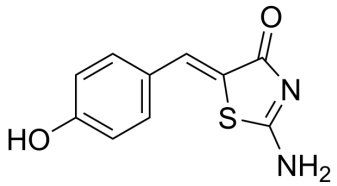
(Z)-Mirin

Cat. No.:	HY-19959
CAS No.:	1198097-97-0
Molecular Formula:	$C_{10}H_8N_2O_2S$
Molecular Weight:	220.25
Target:	ATM/ATR; Apoptosis
Pathway:	Cell Cycle/DNA Damage; PI3K/Akt/mTOR; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 31 mg/mL (140.75 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.5403 mL	22.7015 mL	45.4030 mL
	5 mM	0.9081 mL	4.5403 mL	9.0806 mL
	10 mM	0.4540 mL	2.2701 mL	4.5403 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: ≥ 1 mg/mL (4.54 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (4.54 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (4.54 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

(Z)-Mirin is an MRN (Mre11-Rad50-Nbs1) inhibitor. (Z)-Mirin prevents MRN-dependent activation of ATM without affecting ATM protein kinase activity. (Z)-Mirin inhibits Mre11-associated exonuclease activity. (Z)-Mirin increases apoptosis, triggers a G2/M checkpoint and strongly inhibits homology-directed repair (HDR)[1][2].

In Vitro

(Z)-Mirin (50, 100, 500 μM) inhibits ATM-dependent phosphorylation of Nbs1 and Chk2 and the MRN-dependent autophosphorylation of ATM at Ser1981 in response to DNA double-strand breaks (DSBs). (Z)-Mirin (100 μM) inhibits Mre11

nuclease activity[1].
(Z)-Mirin (10-100 μM; 24 h) shows 50% cytotoxicity at 50 μM in HEK293 cells[1].
(Z)-Mirin (10-100 μM) induces a substantial G2 arrest at concentrations of 50 μM and 100 μM[1].
(Z)-Mirin (18 μM; 48 h) increases apoptosis in PEO4 cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cytotoxicity Assay[1]

Cell Line:	Human embryonic kidney (HEK) 293 cells
Concentration:	10, 25, 50, 100 μM
Incubation Time:	24 h
Result:	Had little effect on cell survival at 25 μM and showed 50% cytotoxicity at 50 μM.

Cell Cycle Analysis[1]

Cell Line:	TOSA4 cells
Concentration:	10, 25, 50, 100 μM
Incubation Time:	
Result:	Induced a substantial G2 arrest at concentrations of 50 μM and 100 μM.

Apoptosis Analysis[2]

Cell Line:	PEO4 cells
Concentration:	18 μM
Incubation Time:	48 h
Result:	Increased apoptosis.

CUSTOMER VALIDATION

- Nat Commun. 2023 Apr 3;14(1):1838.
- Cell Rep. 2022 Nov 15;111716.
- EMBO Rep. 2020 Aug 5;21(8):e48920.
- Neoplasia. 2021 Apr 27;23(5):515-528.
- Utrecht University. 2023 Feb.

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REFERENCES

[1]. Aude Dupré, et al. A forward chemical genetic screen reveals an inhibitor of the Mre11-Rad50-Nbs1 complex. Nat Chem Biol. 2008 Feb;4(2):119-25.

[2]. Adel Alblihy, et al. Selective Killing of BRCA2-Deficient Ovarian Cancer Cells via MRE11 Blockade. Int J Mol Sci. 2023 Jun 30;24(13):10966.

[3]. Rozier L, et al. The MRN-CtIP pathway is required for metaphase chromosome alignment. Mol Cell. 2013 Mar 28;49(6):1097-107.

[4]. Lee JH, et al. Ataxia telangiectasia-mutated (ATM) kinase activity is regulated by ATP-driven conformational changes in the Mre11/Rad50/Nbs1 (MRN) complex. J Biol Chem. 2013 May 3;288(18):12840-51.

[5]. Garner KM, et al. Corrected structure of Mirin, a small-molecule inhibitor of the Mre11-Rad50-Nbs1 complex. Nat Chem Biol. 2009 Mar;5(3):129-30.

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