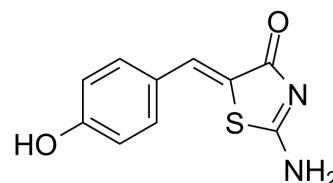


(Z)-Mirin

Cat. No.:	HY-19959
CAS No.:	1198097-97-0
Molecular Formula:	C ₁₀ H ₈ N ₂ O ₂ S
Molecular Weight:	220
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 (140.91 mM)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		4.5455 mL	22.7273 mL	45.4545 mL
	5 mM		0.9091 mL	4.5455 mL	9.0909 mL
	10 mM		0.4545 mL	2.2727 mL	4.5455 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 1 mg/mL (4.55 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1 mg/mL (4.55 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 1 mg/mL (4.55 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 μ M; 24 h) shows 50% cytotoxicity at 50 μ M in HEK293 cells^[1].

(Z)-Mirin (10-100 μ M) induces a substantial G2 arrest at concentrations of 50 μ M and 100 μ M^[1].

(Z)-Mirin (18 μ M; 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 μ M
Incubation Time:	24 h
Result:	Had little effect on cell survival at 25 μ M and showed 50% cytotoxicity at 50 μ M.

Cell Cycle Analysis^[1]

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

Apoptosis Analysis^[2]

Cell Line:	PEO4 cells
Concentration:	18 μ M
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.

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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.

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

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