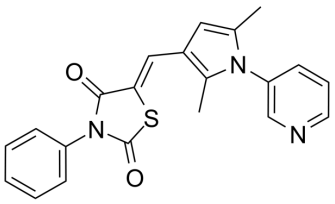
iCRT 14

Cat. No.:	HY-16665
CAS No.:	677331-12-3
Molecular Formula:	$C_{21}H_{17}N_3O_2S$
Molecular Weight:	375.44
Target:	Wnt
Pathway:	Stem Cell/Wnt
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 : ≥ 29 mg/mL (77.24 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6635 mL	13.3177 mL	26.6354 mL
	5 mM	0.5327 mL	2.6635 mL	5.3271 mL
	10 mM	0.2664 mL	1.3318 mL	2.6635 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: ≥ 2.5 mg/mL (6.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

iCRT 14 is a novel potent inhibitor of β-catenin-responsive transcription (CRT), with IC_{50} of 40.3 nM against Wnt responsive STF16 luciferase.

IC_{50} & Target

IC50: 40.3 nM (Wnt responsive STF16 luciferase)[1]

In Vitro

iCRT14 can interfere with TCF binding to DNA in addition to its ability to influence TCF-β-cat interaction[1]. iCRT14 (10, 25, 50 μM) effectively inhibits cell proliferation in BT-549 cells in a dose- and time-dependent manner, but still less potent than iCRT3[2].

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

In Vivo

iCRT14 (50 mg/kg, i.p.) markedly decreases CycD1, proliferation of the tumors in HCT116 xenografts[1].

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PROTOCOL

Cell Assay [2]

Cells are seeded at 20,000 cells/well into 96-well plates. After overnight incubation, cells are treated with DMSO or each Wnt inhibitor (iCRT-3, 75 μM; iCRT-5, 200 μM; iCRT-14, 50 μM; IWP-4, 5 μM and XAV-939, 10 μM) for 48 hours. Cell viability is determined using the Cell Titer-Glo luminescent cell viability assay kit. Luminescence is measured using FLUOstar microplate reader. All treatments are performed in triplicate, and each experiment is repeated three times.

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

CUSTOMER VALIDATION

- Cancer Lett. 2020 Jan 28;469:390-398.
- Cell Death Dis. 2018 Apr 1;9(4):433.
- Elife. 2023 Jan 16;12:e81438.
- Front Bioeng Biotechnol. 2023 Jul 28;11:1215233.
- Neurotherapeutics. 2021 Jan;18(1):601-614.

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REFERENCES

[1]. Gonsalves FC, et al. An RNAi-based chemical genetic screen identifies three small-molecule inhibitors of the Wnt/wingless signaling pathway. Proc Natl Acad Sci USA. 2011 Apr 12;108(15):5954-63.

[2]. Bilir B, et al. Wnt signaling blockage inhibits cell proliferation and migration, and induces apoptosis in triple-negative breast cancer cells. J Transl Med. 2013 Nov 4;11:280.

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