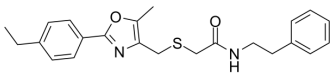
iCRT3

Cat. No.:	HY-103705
CAS No.:	901751-47-1
Molecular Formula:	$C_{23}H_{26}N_2O_2S$
Molecular Weight:	394.53
Target:	Wnt; Apoptosis
Pathway:	Stem Cell/Wnt; 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 : 150 mg/mL (380.20 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5347 mL	12.6733 mL	25.3466 mL
5 mM	0.5069 mL	2.5347 mL	5.0693 mL
10 mM	0.2535 mL	1.2673 mL	2.5347 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 (6.34 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

iCRT3 is an inhibitor of both Wnt and β-catenin-responsive transcription.

IC_{50} & Target

Wnt[1], β-catenin-responsive transcription[2]

In Vitro

iCRT3 is an inhibitor of both Wnt and β-catenin-responsive transcription. iCRT3 significantly decreases TOP Flash activity and reduces the level of NTSR1. The anti-apoptotic effects of Neurotensin (NTS) and Wnt3a can be largely abrogated by iCRT3[1]. Cells maintained long term with iCRT3 show enhanced expression of classic pluripotency genes compare with the DMSO control, whereas expression of differentiation markers and T-cell factor (TCF) target genes is concomitantly reduced [2]. Treatment with iCRT3 at doses of 12.5, 25, 50, and 75 μM decreases TNF-α levels by 14.7%, 18.5%, 44.9% and 61.3%, respectively. With iCRT3 treatment, IκB levels are increased in a dose-dependent manner compare to the vehicle[3].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The tumor growth rates are markedly retarded by iCRT3 treatment. Consistently, the tumor-suppressive role of iCRT3 is accompanied with a reduction in Ki67 index, a proliferation marker[1]. The IL-6 levels in the 10 mg/kg iCRT3 treatment group are 82.9% lower than those in the vehicle group. IL-1β levels are undetectable in the sham but reach 371 pg/mL in septic mice and are down by 30.2% and 53.2%, respectively, with 5 and 10 mg/kg iCRT3. With iCRT3 treatment at doses of 5 and 10 mg/kg, AST levels in these septic mice are 15.4% and 44.2% lower, respectively, than those in the vehicle-treated mice. After treatment with 10 mg/kg iCRT3, lung morphology is improved with much reduced microscopic deterioration, compare to the vehicle group. The number of apoptotic cells in the lung tissues of the iCRT3-treated mice is significantly reduced by 92.7% in comparison with the vehicle group[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cells are seeded into 96-well plates to a density of 5×10^3 cells per well and incubated in the culture medium with iCRT3 for an additional 48 h. Cell viability and cell apoptosis assays are carried out using a Cell Counting kit-8 and a Caspase-Glo 3/7 assay kit according to the manufacturer's instructions, respectively[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	NOD-SCID BALB/c mice are inoculated subcutaneously in the right back with 2×10^6 A172 cells. The growth of the primary tumors is recorded every 4 days. iCRT3 (5 mg/kg) is diluted in PBS i.p. triweekly when tumors grow to ~200 mm^3. The control mice are treated with blank PBS containing 5% (v/v) DMSO. Tumor volume is evaluated with the following formula: volume=tumor length×$width^2$/2. The mice are sacrificed 24 days after pharmaceutical treatment. The tumors are resected and embedded in paraffin, and the Ki67 staining is analyzed by immunohistochemistry[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2022 Nov 2;13(1):6552.
- Nat Commun. 2022 Jul 28;13(1):4364.
- Nat Commun. 2021 Nov 24;12(1):6831.
- Acta Pharm Sin B. 2023 Feb 28.
- BMC Biol. 2023 May 4;21(1):100.

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REFERENCES

[1]. Xiao H, et al. A Novel Positive Feedback Loop Between NTSR1 and Wnt/β-Catenin Contributes to Tumor Growth of Glioblastoma. Cell Physiol Biochem. 2017 Oct 24;43(5):2133-2142.

[2]. Chatterjee SS, et al. Inhibition of β-catenin-TCF1 interaction delays differentiation of mouse embryonic stem cells. J Cell Biol. 2015 Oct 12;211(1):39-51.

[3]. Sharma A, et al. Mitigation of sepsis-induced inflammatory responses and organ injury through targeting Wnt/β-catenin signaling. doi: 10.1038/s41598-017-08711-6.

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

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