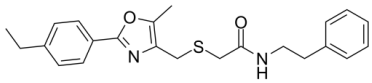
Data Sheet

Product Name:	iCRT3
Cat. No.:	CS-0033387
CAS No.:	901751-47-1
Molecular Formula:	C23H26N2O2S
Molecular Weight:	394.53
Target:	Apoptosis; Wnt
Pathway:	Apoptosis; Stem Cell/Wnt
Solubility:	DMSO : 150 mg/mL (380.20 mM; Need ultrasonic and warming)



BIOLOGICAL ACTIVITY:

iCRT3 is an inhibitor of both **Wnt** and **β-catenin-responsive transcription**. IC50 & 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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

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]. **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].

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.

CAIndexNames:

Acetamide, 2-[[[2-(4-ethylphenyl)-5-methyl-4-oxazolyl]methyl]thio]-N-(2-phenylethyl)-

SMILES:

O=C(NCCC1=CC=CC=C1)CSCC2=C(C)OC(C3=CC=C(CC)C=C3)=N2

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