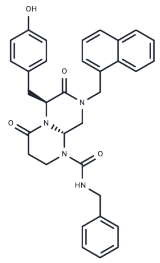
Data Sheet (Cat.No.T6113)

TargetMol

ICG-001

Chemical Properties



CAS No. : 780757-88-2

Formula: C33H32N4O4

Molecular Weight: 548.63

Storage: Keep away from moisture
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	ICG001 is a β-catenin/TCF-mediated transcription inhibitor that selectively blocks β-catenin/CBP interaction without interfering with β-catenin/p300 interaction. It specifically binds to CREB protein with an IC50 of 3 μM.
Targets(IC50)	Apoptosis,Epigenetic Reader Domain,Wnt/beta-catenin
In vitro	**METHODS**: SW480 cells were treated with ICG-001 (25 μM, 24 h) and the inhibition of survivin gene transcription was measured by semiquantitative RT-PCR; SW480 and HCT116 cells were treated with ICG-001 (10 and 25 μM, 24 h) and analyzed by immunoblotting for survivin; SW480 cells were treated with ICG-001 (25 μM) and the chromatin immunoprecipitation assay of the cyclin D1 promoter was used; promoter occupancy was assessed with CBP- (AC-22) or p300- (C-20) specific antibodies in the presence of ICG-001 (25 μM, 8 h). **RESULTS** After 8 h of treatment of SW480 cells with 25 μM ICG-001, approximately 2% of the genes analyzed were upregulated by a factor greater than 2, while only approximately 0.3% of the genes were downregulated by more than 50%. The two genes downregulated were S100A4 and survivin, which are the first and fourth mRNAs upregulated in cancer cells, respectively. ICG-001 effectively reduced the steady-state levels of survivin mRNA and protein in treated colorectal cancer cells. SW480 cells treated with ICG-001 (25 μM) showed a significant decrease in cyclin D1 as early as 4 h after treatment. Using chromatin immunoprecipitation, ICG-001 was shown to selectively inhibit the endogenous cyclin D1 promoter β[1].
In vivo	**METHODS**: In a tumor regression SW620 nude mouse xenograft model, ICG-001 (150 mg/kg, intravenous injection) was administered to observe the effect of the drug on tumor remission in nude mice. **RESULTS** Tumor volume was significantly reduced during the 19-day treatment, with no mortality or weight loss. [1]

Solubility Information

Solubility	DMSO: 242 mg/mL (441.1 mM),Sonication is recommended. Ethanol: 9 mg/mL (16.4 mM),Sonication is recommended. H2O: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)

In vivo Formulation	2% DMSO+40% PEG300+5% Tween 80+53% Saline: 10 mg/mL (18.23 mM),Solution. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.8227 mL	9.1136 mL	18.2272 mL
5 mM	0.3645 mL	1.8227 mL	3.6454 mL
10 mM	0.1823 mL	0.9114 mL	1.8227 mL
50 mM	0.0365 mL	0.1823 mL	0.3645 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Emami KH, et al. A small molecule inhibitor of beta-catenin/CREB-binding protein transcription [corrected]. Proc Natl Acad Sci U S A. 2004 Aug 24;101(34):12682-7.

Miao H, Wang Y, Su W, et al.Sirtuin 6 protects against podocyte injury by blocking the renin-angiotensin system by inhibiting the Wnt1/β-catenin pathway.Acta Pharmacologica Sinica.2023: 1-13.

Yu Z, Gao R, Lv C, et al.Notoginsenoside R1 promotes Lgr5+ stem cell and epithelium renovation in colitis mice via activating Wnt/β-Catenin signaling.Acta Pharmacologica Sinica.2024: 1-15.

Teo JL, et al. Specific inhibition of CBP/beta-catenin interaction rescues defects in neuronal differentiation caused by a presenilin-1 mutation. Proc Natl Acad Sci U S A. 2005 Aug 23;102(34):12171-6.