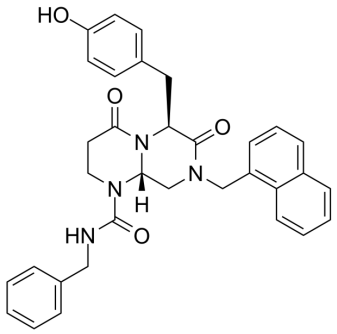
ICG-001



Cat. No.:	HY-14428
CAS No.:	780757-88-2
Molecular Formula:	$C_{33}H_{32}N_4O_4$
Molecular Weight:	548.63
Target:	β-catenin; Apoptosis
Pathway:	Stem Cell/Wnt; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (91.14 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
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

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 (4.56 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.56 mM); Suspended solution; Need ultrasonic and warming
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ICG-001 is an inhibitor of β-catenin/TCF mediated transcription. ICG-001 works by specifically binding to cyclic AMP response element-binding protein with an IC_{50} of 3 μM. ICG-001 selectively blocks the β-catenin/CBP interaction without interfering with the β-catenin/p300 interaction.

IC_{50} & Target

IC50: 3 μM (CBP)

In Vitro	ICG-001 (5μM) inhibits leptin-induced EMT, invasion and tumorsphere formation in MCF7 cells[1]. ICG-001 can phenotypically rescue normal nerve growth factor (NGF)-induced neuronal differentiation and neurite outgrowth in the presenilin-1 mutant cells, emphasizing the importance of the TCF/β-catenin signaling pathway on neurite outgrowth and neuronal differentiation[2]. ICG-001 (25μM) treatment reduces the steady-state levels of Survivin and Cyclin D1 RNA and protein in SW480 cells, both of which can be up-regulated by β-catenin. ICG-001 selectively induces apoptosis in transformed cells but not in normal colon cells, and reduces in vitro growth of colon carcinoma cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	ICG-001 (5 mg/kg per day) significantly inhibits beta-catenin signaling in mice, while concurrently preserving the epithelium [2]. Administration of a water-soluble analog of ICG-001 for 9 weeks reduces the formation of colon and small intestinal polyps by 42% as effectively as the nonsteroidal antiinflammatory agent MK-231, which has consistently demonstrated efficacy in this model. ICG-001 (150 mg/kg, i.v.) demonstrates a dramatic reduction in tumor volume over the 19-day course of treatment, with no mortality or weight loss in the SW620 nude mouse xenograft model of tumor regression[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	To evaluate effects of ICG-001 on α-SMA and collagen type 1 expression, RLE-6TN cells are treated with TGF-β1 (0.25 ng/mL) in the presence or absence of ICG-001 (5.0 μM). After 24 h, cells are harvested and mRNA isolated for analysis by qPCR. RNA is reverse-transcribed using SuperScript reverse transcriptase. Quantitative PCR is performed with SYBR-Green PCR using Real-Time PCR System HT7900. The amplification protocol is set as follows: 95°C denaturation for 10 min followed by 40 cycles of 15-s denaturation at 95°C, 1 min of annealing/extension, and data collection at 60°C. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Seven-week-old male C57BL/6J-Apc$^{Min/+}$and WT C57BL/6J mice are treated orally for 9 weeks with ICG-001a (300 mg/kg per day) or vehicle (1% carboxymethylcellulose), once daily, six times per week. MK-231 is administered in drinking water (160 ppm, dissolved in 8 mM Na_2PO_4 buffer, pH 7.6). At 16 weeks, the polyp number is counted manually by using a dissecting microscope. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Pineal Res. 2019 Sep;67(2):e12588.
- Cancer Res. 2019 Aug 15;79(16):4160-4172.
- Acta Pharm Sin B. 26 October 2021.
- J Exp Clin Cancer Res. 2017 Sep 11;36(1):125.
- Cell Death Dis. 2018 Jul 18;9(8):793.

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REFERENCES

[1]. Yan D, et al, Leptin-induced epithelial-mesenchymal transition in breast cancer cells requires β-catenin activation via Akt/GSK3- and MTA1/Wnt1 protein-dependent pathways. J Biol Chem, 2012, 287(11), 8598-8612.

[2]. Henderson WR Jr, et al, Inhibition of Wnt/beta-catenin/CREB binding protein (CBP) signaling reverses pulmonary fibrosis. Proc Natl Acad Sci USA, 2010, 107(32), 14309-14314.

[3]. Emami KH, et al. A small molecule inhibitor of beta-catenin/CREB-binding protein transcription [corrected]. Proc Natl Acad Sci USA, 2004, 101(34), 12682-12687.

[4]. Liu Y, et al. ICG-001 suppresses growth of gastric cancer cells and reduces chemoresistance of cancer stem cell-like population. J Exp Clin Cancer Res. 2017 Sep 11;36(1):125.

India Contact:
Life Technologies (India) Pvt. Ltd.
306, Aggarwal City Mall, Opposite M2K Pitampura, Delhi – 110034 (INDIA). Ph: +91-11-42208000, 42208111, 42208222, Mobile: +91-9810521400, Fax: +91-11-42208444
Email: customerservice@lifetechindia.com Website: www.lifetechindia.com