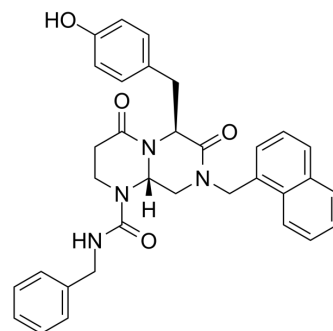


ICG-001

Cat. No.:	HY-14428		
CAS No.:	780757-88-2		
Molecular Formula:	C ₃₃ H ₃₂ N ₄ O 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.

Concentration	Solvent	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- 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
- 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 IC₅₀ of 3 μM. ICG-001 selectively blocks the β-catenin/CBP interaction without interfering with the β-catenin/p300 interaction.

IC₅₀ & Target

IC₅₀: 3 μM (CBP)

In Vitro	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

PROTOCOL

Cell Assay ^[2]	<p>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.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration ^[3]	<p>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 Na₂PO₄ buffer, pH 7.6). At 16 weeks, the polyp number is counted manually by using a dissecting microscope.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

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.

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

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