

Product Data Sheet

Product Name: Wnt-C59

Cat. No.: GC13658

Chemical Properties

Cas. No. 1243243-89-1

Chemical Name 2-[4-(2-methylpyridin-4-yl)phenyl]-N-(4-pyridin-3-ylphenyl)acetamide

SMILES CC1=NC=CC(=C1)C2=CC=C(C=C2)CC(=O)NC3=CC=C(C=C3)C4=CN=CC=C4

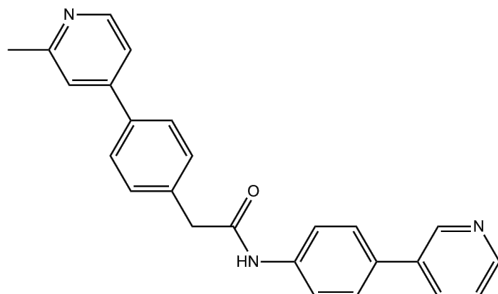
Formula $C_{25}H_{21}N_3O$ M.Wt 379.45

Solubility $\geq 18.95\text{mg/mL}$ in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines NIH 3T3 fibroblasts

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

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Preparation Method	NIH 3T3 fibroblasts were seeded in 6 well plates (50000 cells per well) and treated with Wnt-C59 (0.01 μ M and 1 μ M) for 48h and the expression of genes related to hypertrophy (BNP) and fibrosis (Col1 and Col3) measured in cell lysates.
Reaction Conditions	0.01, 1 μ M; 48h
Applications	Wnt-C59 induced the expression of BNP and both Col1 and Col3 in Fibroblasts.
Animal experiment [2]:	
Animal models	C57BL/6J mice
Preparation Method	Mice were divided into 4 groups: sham, sham+Wnt-C59, transverse aortic constriction (TAC), and TAC+Wnt-C59. Briefly, mice were anesthetized with isoflurane (2%) and a sternotomy was performed to visualize the aortic arch. Then, 7-0 polypropylene suture was used to ligate the aortic arch with a 27G needle as a space holder, which was subsequently removed. The same surgical procedures except for the ligation of the aorta were performed to animals in the sham group. Wnt-C59 was dissolved in saline and administered daily by oral gavage (5mg/kg/day) for experimental groups for 4 weeks. Cardiac function was measured by transthoracic echocardiography using GE vivid 8-dimension ultrasound 4 weeks after TAC surgery. Then mice were anesthetized by isoflurane overdose and hearts were harvested.
Dosage form	5mg/kg; for 4 weeks; p.o.

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Applications

Wnt-C59 significantly improved cardiac function and enhanced survival of mice subjected to TAC surgery. Histologically, Wnt-C59 attenuated TAC-induced increase in heart mass, cross-section area of cardiomyocyte, cardiac fibrosis, cardiomyocyte apoptosis, and expression of the hypertrophic biomarkers β -MHC, ANP, and BNP.

References:

- [1]Paul M A, Wainwright C L, Hector E E, et al. Short-Term Oral Administration of the Porcupine Inhibitor, Wnt-c59, Improves the Structural and Functional Features of Experimental HFpEF[J]. Pharmacology Research & Perspectives, 2025, 13(1): e70054.
- [2]Zhao Z, Liu H, Li Y, et al. Wnt-C59 attenuates pressure overload-induced cardiac hypertrophy via interruption of wnt pathway[J]. Medical Science Monitor: International Medical Journal of Experimental and Clinical Research, 2020, 26: e923025-1.

Background

Wnt-C59 is a highly potent inhibitor of Porcupine (Porcn), a member of the Mboat (membrane-bound O-acyl deglycosylase) family, with an IC_{50} value of 74pM, and can inhibit Wnt signaling^[1]. The Wnt signaling pathway is a complex network of proteins whose functions are most commonly seen in embryonic development and cancer, but are also involved in normal physiological processes in adult animals^[2]. Wnt-C59 has the potential to eradicate cancer stem cells (CSCs) in human tumors^[3]. Wnt-C59 can be used to induce human pluripotent stem cells (IPSCs) to differentiate into cortical motor neurons (CMNs) or their progenitors^[4].

In vitro, Wnt-C59 (0.01, 1 μ M) treatment of NIH 3T3 fibroblasts for 48h significantly induced the expression of genes related to hypertrophy (BNP) and fibrosis (COL1 and COL3) in the cells^[5]. Wnt-C59 (10 μ M) treatment of human fetal liver progenitor cells (HFLPC) significantly inhibited cell migration and proliferation induced by Wnt3a and Wnt5a^[6].

In vivo, Wnt-C59 (5mg/kg) was orally treated for 4 weeks in cardiac hypertrophy model mice, which significantly improved the cardiac function and survival rate of mice, and attenuated the transverse aortic constriction (TAC)-induced cardiac mass, increase in cardiomyocyte cross-sectional area,

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cardiac fibrosis, cardiomyocyte apoptosis, and expression of hypertrophic biomarkers β -MHC, ANP, and BNP^[7]. Wnt-C59 (40, 60mg/kg) was intraperitoneally treated in mice with endotoxin-induced sepsis, which significantly improved the survival rate of mice and reduced the lethality and plasma levels of proinflammatory cytokines and organ damage biomarkers^[8].

References:

- [1] Shah K, Panchal S, Patel B. Porcupine inhibitors: Novel and emerging anti-cancer therapeutics targeting the Wnt signaling pathway[J]. Pharmacological research, 2021, 167: 105532.
- [2] Anastas J N, Moon R T. WNT signalling pathways as therapeutic targets in cancer[J]. Nature Reviews Cancer, 2013, 13(1): 11-26.
- [3] Cheng Y, Phoon Y P, Jin X, et al. Wnt-C59 arrests stemness and suppresses growth of nasopharyngeal carcinoma in mice by inhibiting the Wnt pathway in the tumor microenvironment[J]. Oncotarget, 2015, 6(16): 14428.
- [4] Motono M, Ioroi Y, Ogura T, et al. WNT-C59, a small-molecule WNT inhibitor, efficiently induces anterior cortex that includes cortical motor neurons from human pluripotent stem cells[J]. Stem cells translational medicine, 2016, 5(4): 552-560.
- [5] Paul M A, Wainwright C L, Hector E E, et al. Short-Term Oral Administration of the Porcupine Inhibitor, Wnt-c59, Improves the Structural and Functional Features of Experimental HFpEF[J]. Pharmacology Research & Perspectives, 2025, 13(1): e70054.
- [6] Liu Z, Kuna V K, Xu B, et al. Wnt ligands 3a and 5a regulate proliferation and migration in human fetal liver progenitor cells[J]. Translational Gastroenterology and Hepatology, 2021, 6.
- [7] Zhao Z, Liu H, Li Y, et al. Wnt-C59 attenuates pressure overload-induced cardiac hypertrophy via interruption of wnt pathway[J]. Medical Science Monitor: International Medical Journal of Experimental and Clinical Research, 2020, 26: e923025-1.
- [8] Jang J, Song J, Sim I, et al. Wnt-Signaling Inhibitor Wnt-C59 suppresses the cytokine upregulation in multiple organs of lipopolysaccharide-induced endotoxemic mice via reducing the interaction between β -Catenin and NF- κ B[J]. International Journal of Molecular Sciences, 2021, 22(12): 6249.

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