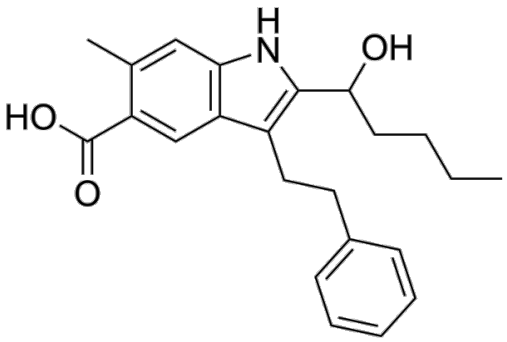
# Product Data Sheet

Product Name: FJ9
Cat. No.: GC79549

## Chemical Properties

| | | | |
|---|---|---|---|
| Cas. No. | 873841-43-1 | | |
| Formula | $C_{23}H_{27}NO_3$ | M.Wt | 365.47 |
| Solubility | DMSO: 100 mg/mL (273.62 mM; Need ultrasonic) | 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




## Background

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FJ9 is a NHERF1/PDZ inhibitor with human NHERF1 PDZ1 $IC_{50}$ 1540 μM, NHERF1 PDZ2 $IC_{50}$ 160 μM, and Frizzled-7-Dishevelled PDZ complex K i 10 μM. FJ9 binds ligand-binding pockets of NHERF1 PDZ domains to block cognate ligand interactions, disrupts Frizzled-7-Dishevelled interactions, and down-regulates canonical Wnt signaling. FJ9 induces apoptosis in cancer cells with intact β-catenin signaling. FJ9 can be used for the research of non-small cell lung cancer, melanoma [1] [2].

In Vivo, FJ9 (50 mg/kg; i.p.; daily; 14 days) significantly inhibits H460 non-small cell lung cancer xenograft growth in nude mice without causing significant weight loss[2].

In Vitro, FJ9 (Compound 1) inhibits the interaction between GST-fused human NHERF1 PDZ1 domain and biotinylated β2AR carboxy-terminal peptide with an $IC_{50}$ of 1540 μM[1]. FJ9 inhibits the interaction between GST-fused human NHERF1 PDZ2 domain and biotinylated CFTR carboxy-terminal peptide with an $IC_{50}$ of 160 μM[1]. FJ9 (0.1-1000 μM) competitively inhibits Frz7 peptide binding to purified human DVL1 PDZ domain with a Ki of 63 μM and to purified human DVL3 PDZ domain with a Ki of 29 μM[2]. FJ9 (100-300 μM; 24 hours) penetrates HEK293T cells and disrupts the intracellular interaction between HA-tagged mouse DVL1 and myc-tagged human Frz7 at concentrations of 100 μM and 300 μM[2]. FJ9 (100-300 μM; 48 hours) dose-dependently down-regulates cytosolic β-catenin levels in hTERT-immortalized LP9 mesothelial cells stably expressing full-length mouse DVL1, but not in control LP9 cells or LP9 cells expressing PDZ domain-deleted mouse DVL1[2]. FJ9 (100-300 μM; 24 hours) dose-dependently inhibits constitutive Tcf transcriptional activity in HCT116 p53-/- colon cancer cells, reducing TOPflash reporter activity while minimally affecting FOPflash reporter activity[2]. FJ9 (30-100 μM; 1-2 days) down-regulates canonical Wnt signaling target proteins (cyclin D1, c-myc, survivin) and mRNA (cyclin D1, survivin) in HCT116 p53-/- colon cancer cells, and down-regulates survivin protein in NCI-H1703 non-small cell lung cancer cells[2]. FJ9 (30-100 μM; 24 hours) does not affect JNK activation in mDVL1-transfected HeLa cells after 24 hours of treatment at 30 μM and 100 μM[2]. FJ9 (86-100 μM; 3 to 6 days) induces significant apoptosis in LOX melanoma, NCI-H460 non-small cell lung cancer, and NCI-H1703 non-small cell lung cancer cells, but has no apoptotic effect on NCI-H28 mesothelioma cells or normal NHBE and SAEC cells, after 3 to 6 days of treatment at 86 to 100 μM[2].

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

[1]. Mayasundari A, et al. Rational design of the first small-molecule antagonists of NHERF1/EBP50 PDZ domains. Bioorg Med Chem Lett. 2008;18(3):942-945.

[2]. Fujii N, et al. An antagonist of dishevelled protein-protein interaction suppresses beta-catenin-dependent tumor cell growth. Cancer Res. 2007;67(2):573-579.

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