SC144 hydrochloride

Cat. No.: HY-15614A

CAS No.: 917497-70-2

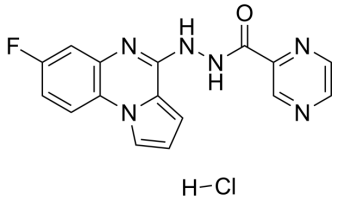
Molecular Formula: $C_{16}H_{12}ClFN_6O$

Molecular Weight: 358.76

Target: Interleukin Related; Apoptosis

Pathway: Immunology/Inflammation; Apoptosis

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10 mg/mL (27.87 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7874 mL	13.9369 mL	27.8738 mL
5 mM	0.5575 mL	2.7874 mL	5.5748 mL
10 mM	0.2787 mL	1.3937 mL	2.7874 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 1 mg/mL (2.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SC144 hydrochloride is a first-in-class, orally active gp130 (IL6-beta) inhibitor. SC144 hydrochloride binds gp130, induces gp130 phosphorylation (S782) and deglycosylation, abrogates Stat3 phosphorylation and nuclear translocation, and further inhibits the expression of downstream target genes. SC144 hydrochloride shows potent inhibition of gp130 ligand-triggered signaling. SC144 hydrochloride induces apoptosis in human ovarian cancer cells[1].

IC_{50} & Target

IL6-beta

In Vitro

SC144 inhibits cell growth in a panel of human ovarian cancer cell lines with IC_{50}s in a submicromolar range (IC_{50}=OVCAR-8, OVCAR-5, OVCAR-3= 0.72, 0.49, 0.95 μM)[1].
The potency of SC144 toward NCI/ADR-RES (Paclitaxel- and Doxorubicin-resistant, IC_{50}=0.43 μM) and HEY (Cisplatin-resistant, IC_{50}=0.88 μM) suggests an ability to overcome drug resistance in ovarian cancer[1].
SC144 (2 μM; 24 hours) causes significantly more apoptosis in OVCAR-8 and Caov-3 than normal kidney epithelial and normal endometrial cells[1].

SC144 (0.5-2 μM; 0-6 hours) substantially increases the phosphorylation of gp130 (S782) in both OVCAR-8 and Caov-3 cells in a time- and dose-dependent manner[1].
SC144 is cytotoxic to ovarian cancer cells via a mechanism involving the inhibition of gp130 activity, leading to the inactivation of Akt and Stat3 as well as the suppression of Stat3-regulated gene expression. As are result, SC144 treatment eventually causes cell-cycle arrest, anti-angiogenesis, and apoptosis[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	OVCAR-8 and Caov-3 cells
Concentration:	2 μM
Incubation Time:	24 hours
Result:	Significantly caused cell death in OVCAR-8 and Caov-3 cells.

Western Blot Analysis[1]

Cell Line:	OVCAR-8, Caov-3 cells
Concentration:	0.5-2 μM
Incubation Time:	0-6 hours
Result:	Substantially increased the phosphorylation of gp130 (S782) in both OVCAR-8 and Caov-3 cellsin a time- and dose-dependent manner.

In Vivo

SC144 (10 mg/kg; i.p.; daily for 58 days) suppresses tumor growth in human ovariancancer xenografts[1].
SC144 (100 mg/kg; p.o.; daily for 35 days) treatment shows the average tumor volume in mice 82% smaller than that in the control group[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Athymic mice (human ovarian cancer xenograft)[1]
Dosage:	I.p; daily for 58 days
Administration:	10 mg/kg
Result:	Significantly inhibited tumor growth by about 73%.

CUSTOMER VALIDATION

- Sci Adv. 2023 Jul 21;9(29):eadh0102.
- Inflamm Res. 2023 Jan 8.
- Biochem Biophys Res Commun. 2023 Oct 10.

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REFERENCES

[1]. Xu S, et al. Discovery of a novel orally active small-molecule gp130 inhibitor for the treatment of ovarian cancer. Mol Cancer Ther. 2013 Jun;12(6):937-49.

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

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