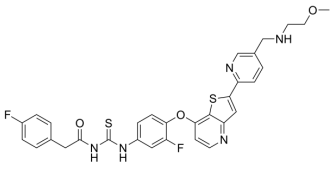
Glesatinib

Cat. No.:	HY-19642
CAS No.:	936694-12-1
Molecular Formula:	$C_{31}H_{27}F_2N_5O_3S_2$
Molecular Weight:	619.7
Target:	TAM Receptor; c-Met/HGFR
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Glesatinib (MGCD265) is an orally active, potent MET/SMO dual inhibitor. Glesatinib, a tyrosine kinase inhibitor, antagonizes P-glycoprotein (P-gp) mediated multidrug resistance (MDR) in non-small cell lung cancer (NSCLC)[1][2].

In Vitro

Glesatinib (MGCD265; 0.01-5 μM; for 72 hours) results in a dose-dependent inhibition of cancer cell growth and shows the low IC_{50} value of 0.08 μM on NSCLC H1299 cells[1].

Glesatinib (0.01, 0.1, 0.5, 1 μM) significantly increases by several-fold the percentage of apoptotic cells in NSCLC H1299 cells [1].

Glesatinib has the cytotoxicity to P-gp overexpressing cancer cells KB-C2, SW620/Ad300, HEK293/ABCB1, and their parent cells KB-3-1, SW620, HEK293 cells with the IC_{50}s fell between 5 and 10 μM[1].

Glesatinib (1, 3 μM; 120 mins) increases the intracellular [^{3}H]-Paclitaxel accumulation and inhibits [^{3}H]-Paclitaxel efflux in cancer cell lines overexpressing P-gp[2].

Glesatinib (0-40 μM) stimulates the ATPase activity of P-gp transporters in a dose-dependent manner[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	NSCLC H1299 cells
Concentration:	0.01, 0.1, 1, 2, 5 μM
Incubation Time:	For 72 hours
Result:	Resulted in a dose-dependent inhibition of cancer cell growth and showed the lowest IC_{50} value of 0.08 μM.

In Vivo

Glesatinib (MGCD265; 15 mg/kg/day; orally; 40 weeks) causes a significant decrease in tumor size[1].

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Animal Model:	4–6-week old female balb/c athymic (nu/nu) mice with HCC827 NSCLC tumor xenografts[1]
Dosage:	15 mg/kg
Administration:	Orally; daily; 40 weeks

Result:	Caused a significant decrease in tumor size.

CUSTOMER VALIDATION

- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Cell Rep. 2022 Dec 13;41(11):111827.

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REFERENCES

[1]. Morgillo F, et al. Dual MET and SMO Negative Modulators Overcome Resistance to EGFR Inhibitors in Human Nonsmall Cell Lung Cancer. J Med Chem. 2017 Sep 14;60(17):7447-7458.

[2]. Cui Q, et al. Glesatinib, a c-MET/SMO Dual Inhibitor, Antagonizes P-glycoprotein Mediated MultidrugResistance in Cancer Cells. Front Oncol. 2019 Apr 25;9:313.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA