

Gilteritinib hemifumarate

Catalog No: tcsc0011363



Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg



Specifications

CAS No:

1254053-84-3

Formula:

$C_{29}H_{44}N_8O_{30.5} \cdot C_4H_4O_4$

Pathway:

Protein Tyrosine Kinase/RTK;Protein Tyrosine Kinase/RTK

Target:

FLT3;TAM Receptor

Purity / Grade:

>98%

Solubility:

DMSO : 1.74 mg/mL (2.85 mM; Need ultrasonic and warming)

Alternative Names:

ASP2215 hemifumarate

Observed Molecular Weight:

610.75

Product Description

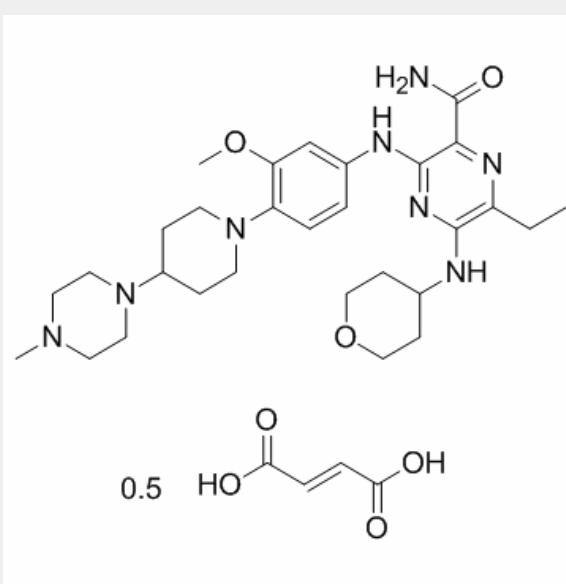
Gilteritinib hemifumarate is a potent **FLT3/AXL** inhibitor with **IC₅₀** of 0.29 nM/0.73 nM, respectively.

IC50 & Target: IC50: 0.29 nM (FLT3)^[1]

IC50: 0.35 nM (LTK), 0.73 nM (AXL), 1.2 nM (EML4-ALK), 230 nM (c-KIT)^[2]

In Vitro: Of the 78 tyrosine kinases tested, Gilteritinib (ASP2215) inhibits FLT3, leukocyte tyrosine kinase (LTK), anaplastic lymphoma kinase (ALK), and AXL kinases by over 50% at 1 nM with an IC₅₀ value of 0.29 nM for FLT3, approximately 800-fold more potent than for c-KIT^[1]. Gilteritinib inhibits the activity of eight of the 78 tested kinases by over 50% at concentrations of either 1 nM (FLT3, LTK, ALK, and AXL) or 5 nM (TRKA, ROS, RET, and MER). The IC₅₀s are 0.29 nM for FLT3 and 0.73 nM for AXL. Gilteritinib inhibits FLT3 at an IC₅₀ that is approximately 800-fold more potent than the concentration required to inhibit c-KIT (230 nM). The antiproliferative activity of Gilteritinib is evaluated against MV4-11 and MOLM-13 cells, which endogenously express FLT3-ITD. After 5 days of treatment, Gilteritinib inhibits the growth of MV4-11 and MOLM-13 cells with mean IC₅₀s of 0.92 nM (95% CI: 0.23-3.6 nM) and 2.9 nM (95% CI: 1.4-5.8 nM), respectively. Growth suppression of MV4-11 cells is accompanied by inhibition of FLT3 phosphorylation. Relative to vehicle control cells, phosphorylated FLT3 levels are 57%, 8%, and 1% after 2 h of treatment with 0.1 nM, 1 nM, and 10 nM Gilteritinib, respectively. In addition, doses as low as 0.1 nM or 1 nM result in the suppression of phosphorylated ERK, STAT5, and AKT, all of which are downstream targets of FLT3 activation. To investigate the effects of Gilteritinib on AXL inhibition, MV4-11 cells that expressed exogenous AXL are treated with Gilteritinib. At concentrations of 1 nM, 10 nM, and 100 nM for 4 h, Gilteritinib treatment decreases phosphorylated AXL levels by 38%, 29%, and 22%, respectively^[2].

In Vivo: In MV4-11 xenografted-mice, the concentration of Gilteritinib (ASP2215) in tumors is more than 20-fold higher than that in plasma with oral administration of Gilteritinib at 10 mg/kg for 4 days. Treatment of Gilteritinib for 28 days results in dose-dependent inhibition of MV4-11 tumor growth and induces complete tumor regression at more than 6 mg/kg. Further, Gilteritinib decreases tumor burden in bone marrow and prolonged the survival of mice intravenously transplanted with MV4-11 cells^[1].



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