

Product Data Sheet

Product Name: Flumatinib mesylate

Cat. No.: GC13914

Chemical Properties

Cas. No. 895519-91-2

Chemical Name methanesulfonic acid;4-[(4-methylpiperazin-1-yl)methyl]-N-[6-methyl-5-[(4-pyridin-3-yl)pyrimidin-2-yl)amino]pyridin-3-yl]-3-(trifluoromethyl)benzamide

SMILES CC1=C(C=C(C=N1)NC(=O)C2=CC(=C(C=C2)CN3CCN(CC3)C)C(F)(F)F)NC4=NC=CC(=N4)C5=CN=CC=C5.CS(=O)(=O)O

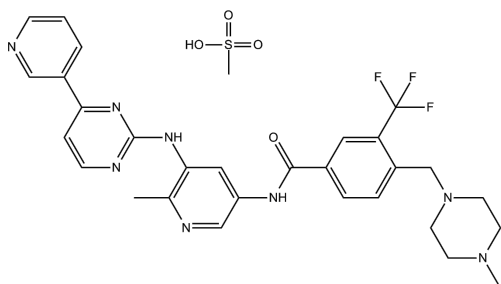
Formula $C_{30}H_{33}F_3N_8O_4S$ M.Wt 658.69

Solubility Soluble 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



Background

Flumatinib mesylate (HH-GV-678 mesylate), a derivative of imatinib, is a multi-kinase inhibitor with IC50 Values of 1.2 nM, 307.6 nM and 2662 nM for c-Abl, PDGFRβ and c-Kit respectively. IC50 Value: 1.2 nM (c-Abl); 307.6 nM (PDGFRβ); 2662 nM (c-Kit) [1]. Target: c-Abl; c-Kit; PDGFRβ. In vitro: HH-GV-678 can predominantly inhibit the autophosphorylation of Bcr-Abl in K562 cell. In higher concentration, HH-GV-678 can inhibit the phosphorylation of c-Kit in Mo7e cell and the phosphorylation of PDGFR in Swiss3T3 cell, however, HH-GV-678 has no or little effect on other tyrosine kinase including EGFR/KDR/c-Src and HER2 [1]. Flumatinib effectively overcame the drug

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

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resistance of certain KIT mutants with activation loop mutations (i.e., D820G, N822K, Y823D, and A829P) [2].in vivo: The purpose of this study was to identify the metabolites of flumatinib in CML patients, with the aim of determining the main metabolic pathways of flumatinib in humans after oral administration. Ultra-performance liquid chromatography/quadrupole time-of-flight mass spectrometry revealed 34 metabolites; 7 primary metabolites were confirmed by comparison with synthetic reference standards. The results show that the parent drug flumatinib was the main form recovered in human plasma, urine, and feces. The main metabolites of flumatinib in humans were the products of N-demethylation, N-oxidation, hydroxylation, and amide hydrolysis [3].

References:

- [1]. Luo H, et al. HH-GV-678, a novel selective inhibitor of Bcr-Abl, outperforms imatinib and effectively overrides imatinib resistance. *Leukemia*. 2010 Oct;24(10):1807-9.
- [2]. Zhao J, et al. Flumatinib, a selective inhibitor of BCR-ABL/PDGFR/KIT, effectively overcomes drug resistance of certain KIT mutants. *Cancer Sci*. 2013 Nov 10.
- [3]. Gong A, et al. Metabolism of flumatinib, a novel antineoplastic tyrosine kinase inhibitor, in chronic myelogenous leukemia patients. *Drug Metab Dispos*. 2010 Aug;38(8):1328-40.

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