Product Datasheet

Avapritinib

Catalog No. **BP-01923** · CAS **1703793-34-3** · Form **free base** · Physical state **solid** · Color **White to light yellow** · Version **1.0** · Revision **2026-09-03**

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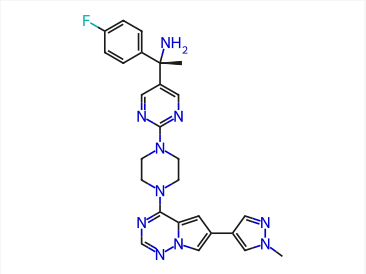
Target	Research area
c-Kit; PDGFR	**Cancer**

Molecular formula	$C_{26}H_{27}FN_{10}$
Molecular weight	**498.5580**

STRUCTURE



DESCRIPTION

Avapritinib (BLU-285) is a highly potent, selective, and orally active KIT and PDGFRA activation loop mutant kinases inhibitor with IC50s of 0.27 and 0.24 nM for KIT D816V and PDGFRA D842V, respectively. Avapritinib (BLU-285) binds the active conformation of the kinase and shows antitumor activity.

BIOLOGICAL ACTIVITY

In Vitro

Avapritinib (BLU-285) has demonstrated biochemical in vitro activity on the KIT exon 17 mutant enzyme, KIT D816V (IC50=0.27 nM). Cellular activity of Avapritinib on KIT D816 mutants is measured by autophosphorylation in the human mast cell leukemia cell line HMC1.2, and the P815 mouse mastocytoma cell line with IC50=4 and 22 nM, respectively. In Kasumi-1 cells, a t(8;21)-positive AML cell line with a KIT exon 17 N822K mutation, Avapritinib potently inhibits KIT N822K mutant autophosphorylation (IC50=40 nM), downstream signaling, as well as cellular proliferation (IC50=75 nM).

In Vivo

In vivo Avapritinib (BLU-285) is well tolerated and has demonstrated dose dependent antitumor efficacy. Complete tumor growth inhibition and ≥75% KIT kinase inhibition is observed with 10 mg/kg once daily, oral dosing of Avapritinib in the aggressive KIT exon 17 mutant driven P815 mastocytoma model grown as a solid tumor allograft as well as in a disseminated model of disease. Disease burden, measured by whole body luciferase imaging (photons/second/mm2), increases 86-fold in the vehicle control animals over the 24 day dosing period with widespread disease detectable in both femurs, the pelvis and circulating in peripheral blood. Avapritinib at both doses (10 or 30 mg/kg orally, once daily) results in a marked reduction of disease burden throughout the study. Avapritinib at either 10 or 30 mg/kg results in tumor regression in all animals with disease abrogation indistinguishable from background signal measurements in several animals by the end of study. Avapritinib is also well tolerated in this in vivo model and has no adverse effects on body weight at either dose.

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Wu CP, et al. Avapritinib: A Selective Inhibitor of KIT and PDGFRα that Reverses ABCB1 and ABCG2-MediatedMultidrug Resistance in Cancer Cell Lines. Mol Pharm. 2019 Jul 1;16(7):3040-3052.

[2]. Evans EK, et al. A precision therapy against cancers driven by KIT/PDGFRA mutations. Sci Transl Med. 2017 Nov 1;9(414). pii: eaao1690.

[3]. Erica Evans, et al. Blu-285, a Potent and Selective Inhibitor for Hematologic Malignancies with KIT Exon 17 Mutations.Blood 2015 126:568.