Product Datasheet

AZD1480

Catalog No. **BP-02218** · CAS **935666-88-9** · Form **free base** · Physical state **solid** · Color **White to yellow** · Version **1.0** · Revision **2026-09-02**

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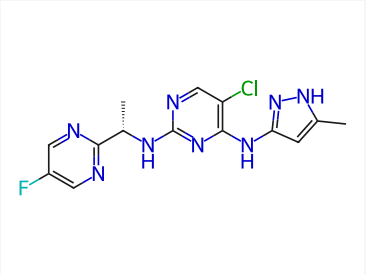
Target	Research area
JAK	**Cancer**

Molecular formula	$C_{14}H_{14}ClFN_8$
Molecular weight	**348.7660**

STRUCTURE



DESCRIPTION

AZD1480 is a novel, potent and selective small-molecule JAK2 inhibitor ((IC50 = 0.26 nM). It can effectively inhibit tumor angiogenesis and metastasis mediated by STAT3 in stromal cells as well as tumor cells. Also demonstrates >50-fold selectivity for JAK2 over JAK3.

BIOLOGICAL ACTIVITY

In Vitro

AZD-1480 (5μM) induces G2/M arrest and cell death by inhibiting Aurora kinases.

AZD-1480 is a potent JAK2 inhibitor that can suppress growth, survival, as well as FGFR3 and STAT3 signaling and downstream targets including Cyclin D2 in human multiple myeloma cells. At low micromolar concentrations, AZD-1480 blocks cell proliferation and induces apoptosis of myeloma cell lines.

AZD-1480 effectively blocks constitutive and stimulus-induced JAK1, JAK2, and STAT-3 phosphorylation in both human and murine glioma cells, and leads to a decrease in cell proliferation and induction of apoptosis.

AZD-1480 is a potent, competitive small-molecule inhibitor of JAK1/2 kinase, and that it is capable of inhibiting STAT3 phosphorylation and tumor growth in a STAT3-dependent manner. AZD-1480 inhibits tumor angiogenesis and metastasis in part by affecting the tumor microenvironment.

In Vivo

AZD-1480 inhibits the STAT3 phosphorylation in an xenograft model of human solid tumors and multiple myeloma.

In vivo, AZD-1480 inhibits the growth of subcutaneous tumors and increases survival of mice bearing intracranial glioblastoma (GBM) tumors by inhibiting STAT-3 activity, indicating that pharmacologic inhibition of the JAK/STAT-3 pathway by AZD-1480 should be considered for study in the treatment of patients with GBM tumors.

AZD-1480 blocks lung infiltration of myeloid cells and formation of pulmonary metastases in both mouse syngeneic experimental and spontaneous metastatic models. Furthermore, AZD-1480 reduces angiogenesis and metastasis in a human xenograft tumor model.

AZD-1480 suppresses the growth of human solid tumor xenografts harboring persistent Stat3 activity.

SOLUBILITY

Soluble in DMSO

In vitro	DMSO : 50 mg/mL

REFERENCES

[1]. Derenzini E, et al. The JAK inhibitor AZD1480 regulates proliferation and immunity in Hodgkin lymphoma. Blood Cancer J. 2011 Dec;1(12):e46.

[2]. Scuto A, et al. The novel JAK inhibitor AZD1480 blocks STAT3 and FGFR3 signaling, resulting in suppression of human myeloma cell growth and survival. Leukemia. 2011 Mar;25(3):538-50.

[3]. McFarland BC, et al. Therapeutic potential of AZD1480 for the treatment of human glioblastoma. Mol Cancer Ther. 2011 Dec;10(12):2384-93.

[4]. Xin H, et al. Antiangiogenic and antimetastatic activity of JAK inhibitor AZD1480. Cancer Res. 2011 Nov 1;71(21):6601-10.

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[6]. Ni J, et al. Tyrosine receptor kinase B is a drug target in astrocytomas. Neuro Oncol. 2017 Jan;19(1):22-30.