Barasertib-HQPA

Cat. No.: HY-10126

CAS No.: 722544-51-6

Molecular Formula: $C_{26}H_{30}FN_7O_3$

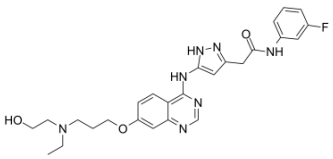
Molecular Weight: 507.56

Target: Aurora Kinase; Apoptosis

Pathway: Cell Cycle/DNA Damage; Epigenetics; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 22 mg/mL (43.34 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9702 mL	9.8511 mL	19.7021 mL
5 mM	0.3940 mL	1.9702 mL	3.9404 mL
10 mM	0.1970 mL	0.9851 mL	1.9702 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.93 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.93 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Barasertib-HQPA (AZD2811) is a highly selective Aurora B inhibitor with an IC_{50} of 0.37 nM in a cell-free assay. Barasertib-HQPA (AZD2811) induces growth arrest and apoptosis in cancer cells[1].

IC_{50} & Target

Aurora B

0.37 nM (IC_{50})

In Vitro

Barasertib-HQPA (3 μM, 3 hours) significantly decreases expression of the phosphorylated forms of histone H3 in freshly isolated leukemia cells[1].

Barasertib-hydroxyquinazoline pyrazol anilide (HQPA)] is converted rapidly to the active Barasertib-HQPA in plasma[2].

Barasertib-HQPA treatment induced defective cell survival, polyploidy, and cell death in LNCaP cell line[3].
Barasertib-HQPA induces a marked anti-propliferative effect accompanied by the appearance of a polyploid population, which in most cases led to apoptosis[4].

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

Cell Proliferation Assay[1].

Cell Line:	AML lines (HL-60, NB4, MOLM13), ALL line (PALL-2), biphenotypic leukemia (MV4-11), acute eosinophilic leukemia (EOL-1), and the blast crisis of chronic myeloid leukemia K562 cells.
Concentration:	0-100 nM. (Barasertib -HQPA)
Incubation Time:	48 h.
Result:	IC_{50} values ranged from 3 nM to 40 nM.

In Vivo

Barasertib (AZD1152, 25 mg/kg) markedly suppresses the growth and weights of AZD1152-treated tumors[1].
Barasertib (AZD1152, 5 mg/kg) enhances the ability of vincristine or daunorubicin to inhibit the proliferation of human MOLM13 leukemic xenografts[1].
Barasertib (AZD1152, (10-150 mg/kg/d) potently inhibited the growth of human colon, lung, and hematologic tumor xenografts (mean tumor growth inhibition range, 55% to z100%; $P < 0.05$) in immunodeficient mice[2].

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

CUSTOMER VALIDATION

- Science. 2017 Dec 1;358(6367):eaan4368.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- J Cell Sci. 2019 Jul 1;132(13):jcs229385.
- Ann Transl Med. 2020 May;8(10):646.
- Research Square Preprint. 2021 Jan.

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REFERENCES

[1]. Wilkinson RW, et al. AZD1152, a selective inhibitor of Aurora B kinase, inhibits human tumor xenograft growth by inducing apoptosis. Clin Cancer Res. 2007 Jun 15;13(12):3682-8.

[2]. Yang, Jing., et al. AZD1152, a novel and selective aurora B kinase inhibitor, induces growth arrest, apoptosis, and sensitization for tubulin depolymerizing agent or topoisomerase II inhibitor in human acute leukemia cells in vitro and in vivo. Blood. 2007 Sep 15;110(6):2034-40.

[3]. Zekri A, et al. AZD1152-HQPA induces growth arrest and apoptosis in androgen-dependent prostate cancer cell line (LNCaP) via producing aneugenic micronuclei and polyploidy. Tumour Biol. 2015 Feb;36(2):623-32.

[4]. Oke A, et al. AZD1152 rapidly and negatively affects the growth and survival of human acute myeloid leukemia cells in vitro and in vivo. Cancer Res. 2009 May 15;69(10):4150-8.

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

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