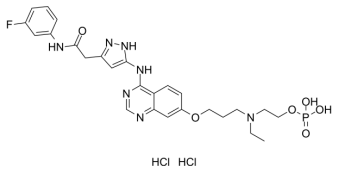
Barasertib dihydrochloride

Cat. No.:	HY-10127A
CAS No.:	722543-50-2
Molecular Formula:	$C_{26}H_{33}Cl_2FN_7O_6P$
Molecular Weight:	660.46
Target:	Aurora Kinase; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Barasertib (AZD1152 dihydrochloride), a pro-drug of Barasertib-hQPA, is a highly selective Aurora B inhibitor with an IC_{50} of 0.37 nM in a cell-free assay. Barasertib (AZD1152 dihydrochloride) induces growth arrest and apoptosis in cancer cells[1].
IC_{50} & Target	IC50: 0.37 nM (Aurora B)
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 induces a marked anti-proliferative effect accompanied by the appearance of a polyploid population, which in most cases led to apoptosis[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Barasertib (AZD1152, 25 mg/kg) markedly suppresses the growth and weights of AZD1152 dihydrochloride-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.
- Nat Commun. 2023 Oct 10;14(1):6332.
- Nat Commun. 2019 Apr 18;10(1):1812
- Dev Cell. 2023 Oct 18:S1534-5807(23)00521-X.
- Clin Cancer Res. 2019 Jul 15;25(14):4552-4566.

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REFERENCES

[1]. Yang J, 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

[2]. 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.

[3]. 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.

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