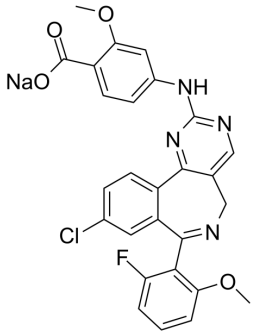
Alisertib sodium

Cat. No.:	HY-10971A
CAS No.:	1028486-06-7
Molecular Formula:	$C_{27}H_{19}ClFN_4NaO_4$
Molecular Weight:	540.91
Target:	Aurora Kinase; Autophagy; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Autophagy; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Alisertib (MLN 8237) sodium is an orally active and selective Aurora A kinase inhibitor (IC_{50}=1.2 nM), which binds to Aurora A kinase resulting in mitotic spindle abnormalities, mitotic accumulation. Alisertib sodium induces apoptosis and autophagy through targeting the AKT/mTOR/AMPK/p38 pathway in leukemic cells. Antitumor activity[1][2][3].

IC_{50} & Target

Aurora A	Aurora B
12.5 nM (IC_{50})	396.5 nM (IC_{50})

In Vitro

Alisertib (MLN 8237) leads the MM cells to mitotic spindle abnormalities, mitotic accumulation, as well as inhibition of cell proliferation through apoptosis and senescence. Alisertib up-regulates p53 and tumor suppressor genes p21 and p27[1].
The decreased activity of Alisertib (MLN 8237) for the T217D/W277E Aurora A/TPX2 complex may reflect the increased affinity for ATP induced by cofactor binding to Aurora A[2].
Alisertib (MLN 8237) inhibits cell proliferation with IC_{50}s ranging from 15 to 469 nM in different tumer cell lines[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Alisertib (MLN 8237) (30 mg/kg, p.o.) significantly reduces tumor burden and increases overall survival in xenograft-murine model of human-MM[1].
Alisertib (3-30 mg/kg; P.o.; once daily for 3 weeks) causes tumor growth inhibition in solid tumor xenograft models[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice bearing HCT-116 colon tumor xenograft[4]
Dosage:	3, 10, or 30 mg/kg
Administration:	P.o.; once daily for 3 weeks
Result:	Resulted in a dose-dependent TGI (tumor growth inhibition) of 43.3%, 84.2%, and 94.7% for the 3, 10, and 30 mg/kg groups,respectively.

CUSTOMER VALIDATION

- Cell. 2017 Jan 12;168(1-2):264-279.e15.

- J Hepatol. 2021 Apr 14;S0168-8278(21)00235-X.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Clin Cancer Res. 2019 Jul 1;25(13):4179-4193.
- Theranostics. 2018 Feb 12;8(6):1740-1751.

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REFERENCES

[1]. Güllü G, et al. A novel Aurora-A kinase inhibitor MLN8237 induces cytotoxicity and cell-cycle arrest in multiple myeloma Blood June 24, 2010 vol. 115 no. 25 5202-5213.

[2]. Sloane DA, et al. Drug-Resistant Aurora A Mutants for Cellular Target Validation of the Small Molecule Kinase Inhibitors MLN8054 and MLN8237 ACS Chem. Biol., 2010, 5 (6), pp 563-576.

[3]. Bavetsias V, et al. Aurora Kinase Inhibitors: Current Status and Outlook. Front Oncol. 2015 Dec 21;5:278.

[4]. Manfredi MG, et al. Characterization of Alisertib (MLN8237), an investigational small-molecule inhibitor of aurora A kinase using novel in vivo pharmacodynamic assays.Clin Cancer Res. 2011 Dec 15;17(24):7614-7624.

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