Product Datasheet

LY2606368

Catalog No. **BP-02309** · CAS **1234015-52-1** · Form **free base** · Physical state **solid** · Color **Light yellow to yellow** · Version **1.0** · Revision **2026-09-10**

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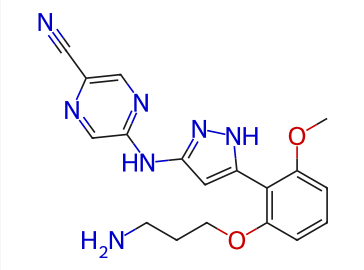
Target	Research area
Checkpoint Kinase (Chk); Apoptosis	**Cancer**

Molecular formula	$C_{18}H_{19}N_7O_2$
Molecular weight	**365.3892**

STRUCTURE



DESCRIPTION

Prexasertib (LY2606368) is a selective, ATP-competitive second-generation checkpoint kinase 1 (CHK1) inhibitor with a Ki of 0.9 nM and an IC50 of <1 nM. Prexasertib inhibits CHK2 (IC50=8 nM) and RSK1 (IC50=9 nM). Prexasertib causes double-stranded DNA breakage and replication catastrophe resulting in apoptosis. Prexasertib shows potent anti-tumor activity.

BIOLOGICAL ACTIVITY

In Vitro

Prexasertib (LY2606368) inhibits MELK (IC50=38 nM), SIK (IC50=42 nM), BRSK2 (IC50=48 nM), ARK5 (IC50=64 nM). LY2606368 requires CDC25A and CDK2 to cause DNA damage.

Prexasertib (33, 100 nM; for 7 hours) results in DNA damage during S-phase in HeLa cells[1].

Prexasertib (8-250 nM; pre-treated for 15 minutes) inhibits CHK1 autophosphorylation (S296) and CHK2 autophosphorylation (S516) in HT-29 cells.

Prexasertib (4 nM; 24 hours) results in a large shift in cell-cycle populations from G1 and G2-M to S-phase with an accompanied induction of H2AX phosphorylation in U-2 OS cells.

Prexasertib (33 nM; for 12 hours) causes chromosomal fragmentation in HeLa cells. Prexasertib (100 nM; 0.5 to 9 hours) induces replication stress and depletes the pool of available RPA2 for binding to DNA.

In Vivo

Prexasertib (LY2606368; 1-10 mg/kg; SC; twice daily for 3 days, rest 4 days; for three cycles) causes growth inhibition in tumor xenografts[1].

Prexasertib (15 mg/kg; SC) causes CHK1 inhibition in the blood and the phosphorylation of both H2AX (S139) and RPA2 (S4/S8).

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. King C, et al. LY2606368 Causes Replication Catastrophe and Antitumor Effects through CHK1-Dependent Mechanisms. Mol Cancer Ther. 2015 Sep;14(9):2004-1

[2]. Yin Y, et al. Chk1 inhibition potentiates the therapeutic efficacy of PARP inhibitor BMN673 in gastric cancer. Am J Cancer Res. 2017 Mar 1;7(3):473-483.