Product Data Sheet

Product Name: Prexasertib Mesylate Hydrate
Cat. No.: GC36967

Chemical Properties

Cas. No.	1234015-57-6		
SMILES	N#CC1=NC=C(NC2=NNC(C3=C(OC)C=CC=C3OCCCN)=C2)N=C1.CS(=O)(O)=O.O		
Formula	$C_{19}H_{25}N_7O_6S$	M.Wt	479.51
Solubility	Soluble in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Prexasertib Mesylate Hydrate		

Background

Prexasertib Mesylate Hydrate (LY2606368 Mesylate Hydrate) is a potent, selective, ATP competitive CHK1 and CHK2 inhibitor, with a Ki of 0.9 nM for CHK1 and IC50s of <1 nM, 8 nM for CHK1 and CHK2, respectively. Prexasertib Mesylate Hydrate inhibits HT-29 CHK1 autophosphorylation (S296) and HT-29 CHK2 autophosphorylation (S516). Prexasertib Mesylate Hydrate shows potent anti-tumor activity, significantly abrogates the G2/M checkpoint in p53 deficient HeLa cells with an EC50 of 9 nM[1]. Chk1|0.9 nM (Ki)|Chk1|<1 nM (IC50)|Chk2|8 nM (IC50)

Prexasertib Mesylate Hydrate (LY2606368 Mesylate Hydrate) is an ATP competitive CHK1 inhibitor, with a Ki of 0.9 nM and an IC50 of <1 nM[1].Prexasertib (LY2606368) shows high anti-tumor activity against U-2 OS, Calu-6 and HeLa cells (IC50, 3, 3, 37 nM, respectively), causes DNA damage during S-phase requiring CDC25A and CDK2 at 4 μM[1].Prexasertib (0-20 nM) synergizes with olaparib (0-20 μM) to decrease cell viability in HGSOC cells[2].

[1]. King C, et al. LY2606368 Causes Replication Catastrophe and Antitumor Effects

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

through CHK1-Dependent Mechanisms. Mol Cancer Ther. 2015 Sep;14(9):2004-13. [2]. Brill E, et al. Prexasertib, a cell cycle checkpoint kinases 1 and 2 inhibitor, increases in vitro toxicity of PARP inhibition by preventing Rad51 foci formation in BRCA wild type high-grade serous ovarian cancer. Oncotarget. 2017 Oct 31;8(67):111026-111040.

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Address: 10292 Central Ave. #205, Montclair, CA, USA