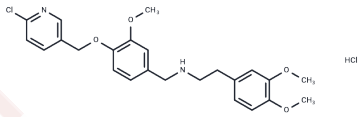
Data Sheet (Cat.No.T2271)

TargetMol

SBE13 Hydrochloride

Chemical Properties



CAS No. : 1052532-15-6

Formula: C24H28Cl2N2O4

Molecular Weight: 479.4

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	SBE13 Hydrochloride (SBE 13 HCl) is an effective and specific PLK1 inhibitor (IC50: 0.2 nM); no inhibition om Aurora A kinase, Plk2/3.
Targets(IC50)	Apoptosis,Autophagy,PLK
In vivo	SBE 13 does not impair the cell cycle or proliferation of primary cells, yet it diminishes the proliferation of various cancer cell lines, induces G2/M arrest, and promotes apoptosis. Co-administration of SBE 13 with Enzastaurin synergistically reduces cell proliferation and enhances apoptosis induction in HCT116 (p53-/-) cells.
Kinase Assay	Kinase assays: To assay Plk1 and Aurora A kinase activity, cells are lysed after 13 hrs release in the presence of SBE13 after double thymidine block, and kinases are immunoprecipitated from lysates using antibodies as described. In brief, for each immunoprecipitation 800 µg of total protein were incubated with 1.5 µg Plk1 antibody cocktail, 3 µg Plk2 antibody, 3 µg Plk3 antibody, or 5 µg Aurora A antibody, respectively, for 2 hrs at 4°C on a rotator. Immunoprecipitated protein is collected using Protein G Agarose beads. The Plk1, Plk2 and Plk3 immunoprecipitates are incubated with 1 µg casein and with 1 µCi of [γ32-P]ATP for 30 min at 37°C in kinase buffer. The Aurora A immunoprecipitates are incubated with 0.5 µl Histone and with 1 µCi of [γ32-P]ATP for 60 min at room temperature in kinase buffer. Products from the kinase assays are fractionated on 10% Bis-Tris-polyacrylamide gels, and the phosphorylated substrate is visualized by autoradiography after an exposure of 12 to 36 hrs. An equal amount of immunoprecipitates is subjected to western blot analysis to confirm equal loading of Plk1, Plk2, Plk3 or Aurora A protein in kinase reactions. Immunoprecipitated Plk1 after 13 hrs release in the presence of SBE13 is assayed after de-phosphorylation using λ protein phosphatase and compared to kinase activity of endogenous immunoprecipitated Plk1. Activity of Plk1 kinase with and wiiiuithout de-phosphorylation is compared and the ratio between de-phosphorylated and "normal" endogenous immunoprecipitated Plk1 kinase activity is calculated.
Cell Research	Cells are treated with SBE13 one day after subculturing. Control cells are incubated with normal culture medium. Concentrations of SBE13 ranged from 1 nM–100 µM. The growth rate of 1 x 105 cells per 6-well is determined by counting cells at 24, 48 and 72 hours after treatment. Cell culture studies are performed in triplicate for each time point.(Only for Reference)

Solubility Information

Solubility	H2O: 4.8 mg/mL (10.01 mM),Heating is recommended. DMSO: 80 mg/mL (166.88 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 3.3 mg/mL (6.88 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.0859 mL	10.4297 mL	20.8594 mL
5 mM	0.4172 mL	2.0859 mL	4.1719 mL
10 mM	0.2086 mL	1.043 mL	2.0859 mL
50 mM	0.0417 mL	0.2086 mL	0.4172 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Keppner S, et al. Cell Cycle. 2010, 9(4), 761-773.
Keppner S, et al. Cell Cycle. 2011, 10(4), 708-720.
Lange L, et al. Oncotarget. 2014, 5(8), 2263-2275.