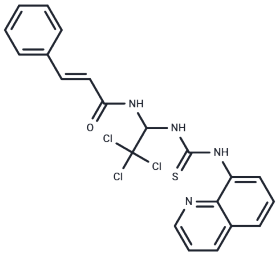
Data Sheet (Cat.No.T3045)

TargetMol

Salubrinal

Chemical Properties



CAS No. : 405060-95-9

Formula: C21H17Cl3N4OS

Molecular Weight: 479.81

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	Salubrinal, a phosphatases (PP1) inhibitor(IC50=1.7 μM), exhibits function on the eukaryotic translation initiation factor 2 subunit (eIF2α).
Targets(IC50)	Apoptosis,Autophagy,HSV,PERK,Phosphatase
In vitro	In a murine model of corneal infection, Salubrinal inhibits HSV replication and lowers viral titers in eye swabs of infected animals. Intraventricular administration of Salubrinal significantly alters the homeostatic sleep response.
In vivo	Salubrinal (EC50=15 μM) inhibits tunicamycin-induced ER stress and subsequent apoptosis in a dose-dependent manner. Additionally, Salubrinal (IC50=3 μM) hampers HSV replication by inhibiting the dephosphorylation of eIF2α. In C12 cells, it downregulates cyclin D1 while upregulating GADD34 and CHOP.
Kinase Assay	Phosphatase activities are determined on immunoprecipitates of the phosphatases. Briefly, 2×106 K562 cells are treated for 18 hr with Salubrinal (20 μM), PSI (10 nM), the combination of both drugs or okadaic acid (100 nM). After washing with PBS, cells are lysed for 15 min on ice either in PP1LB (for determination of PP1γ-activity; 20 mM Tris-HCl, pH 7.5, 1% Triton X-100, 10% glycerol, 132 mM NaCl, Roche complete protease inhibitor) or in RIPA (for PP2A), supplemented with Roche complete protease inhibitor). Cell lysates containing 500 μg (PP1γ) or 300 μg (PP2A) protein are immunoprecipitated overnight at 4°C with 2-3 μg of the appropriate antibodies and then incubated with Protein A-Sepharose. Immunoprecipitates are washed three times in lysis buffer, followed by resuspension in phosphatase assay buffer (PP2A: 20 mM Tris-HCl, pH7.5, 0.1 mM CaCl2; PP1γ: 50 mM Tris HCl pH 7.0, 0.2 mM MnCl2, 0.1 mM CaCl2, 125 μg/mL BSA, 0.05% Tween 20), supplemented with 100 μM 6,8-difluoro-4-methyl-umbelliferyl phosphate (DiFMUP). Precipitates are allowed to react with substrate for 1 hr at 37°C on an Eppendorf Thermoshaker, centrifuged and DiFMU fluorescence is measured on a BioTek Lambda Fluoro 320 microplate reader (360 nmex/460 nmem). Phosphatase activities are given as percent change relative to the control (DMSO treated cells)[1].
Cell Research	PC12 cells are plated in 384-well plates at 5000 cells per well in 40μL phenol red-freemedium containing 3μg/ml Tm to induce ER stress. 100 nL of the DiverSet E (5 mg/mlin DMSO) or National Cancer Institute's (NCI) Structural Diversity set and Open Collections (10 mM in DMSO) (NCI) are added to the wells by robotic pin transfer. After 48 hours, cell viability is assessed using a luminescence-based ATP assay. DMSO- and zVAD.fmk-treated wells on each plate served as negative and positive controls for

Cell Research	rescue from ER stress-induced ATP loss, respectively.(Only for Reference)

Solubility Information

Solubility	DMSO: 40 mg/mL (83.37 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.0842 mL	10.4208 mL	20.8416 mL
5 mM	0.4168 mL	2.0842 mL	4.1683 mL
10 mM	0.2084 mL	1.0421 mL	2.0842 mL
50 mM	0.0417 mL	0.2084 mL	0.4168 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

Boyce M, et al. Science, 2005, 307(5711), 935-939.
Meng C, Zhang J, Dang B, et al. PERK pathway activation promotes intracerebral hemorrhage induced secondary brain injury by inducing neuronal apoptosis both in vivo and in vitro. Frontiers in Neuroscience. 2018 Feb 28;12: 111
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Meng C, Zhang J, Dang B, et al. PERK pathway activation promotes intracerebral hemorrhage induced secondary brain injury by inducing neuronal apoptosis both in vivo and in vitro[J]. Frontiers in neuroscience. 2018 Feb 28;12: 111.