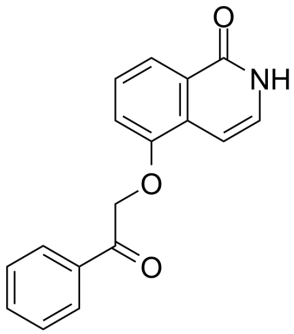
UPF 1069

Cat. No.:	HY-14478
CAS No.:	1048371-03-4
Molecular Formula:	$C_{17}H_{13}NO_3$
Molecular Weight:	279.29
Target:	PARP
Pathway:	Cell Cycle/DNA Damage; Epigenetics
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (358.05 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5805 mL	17.9025 mL	35.8051 mL
5 mM	0.7161 mL	3.5805 mL	7.1610 mL
10 mM	0.3581 mL	1.7903 mL	3.5805 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (8.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

UPF 1069 is a PARP inhibitor, with IC_{50}s of 8 and 0.3 μM for PARP-1 and PARP-2, respectively.

IC_{50} & Target

PARP-2	PARP-1
0.3 μM (IC_{50})	8 μM (IC_{50})

In Vitro

UPF 1069 (Compound 55) is a PARP inhibitor, with IC_{50}s of 8 and 0.3 μM for PARP-1 and PARP-2, respectively[1]. UPF 1069 (1 μM) reduces the residual PARP activity by approximately 80% of PARP-1-deficient fibroblasts, but only slightly inhibits the enzymic activity in wild-type fibroblasts. UPF 1069 (0.1-1 μM) markedly enhances CA1 hippocampal damage. UPF 1069 (10 μM) also exacerbates oxygen-glucose deprivation (OGD) damage in organotypic hippocampal slices. However, UPF 1069

alleviates the damage cuased by OGD in mixed cortical cell cultures, shows a potent neuroprotective activity both at a concentration (1 μM) selectively acting on PARP-2 and at a concentration (10 μM) inhibiting both PARP-1 and PARP-2 activities[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]

PARP activity is evaluated by utilizing commercially available recombinant bovine PARP-1 and mouse PARP-2. Briefly, the enzymatic reaction is carried out in 100 μL of 50 mM Tris-HCl (pH 8.0) containing 5 mM $MgCl_2$, 2 mM dithiothreitol, 10 μg sonicated calf thymus DNA, 0.2 μCi [adenine-2,8-^{3}H]NAD and recombinant enzyme PARP-1 or PARP-2 (0.03 U per sample). Different concentrations of the putative inhibitors are added, and the mixture is incubated for 1 h at 37°C. The reaction is terminated by adding 1 mL of 10% trichloroacetic acid (w/v) and centrifuged. Pellets are then washed twice with 1 mL of H_2O and resuspended in 1 mL of 0.1 M NaOH. The radioactivity incorporated from [adenine-2,8-^{3}H]NAD into proteins is evaluated by liquid scintillation spectrometry[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Pellicciari R, et al. On the way to selective PARP-2 inhibitors. Design, synthesis, and preliminary evaluation of a series of isoquinolinone derivatives. ChemMedChem. 2008 Jun;3(6):914-23.

[2]. Moroni F, et al. Selective PARP-2 inhibitors increase apoptosis in hippocampal slices but protect cortical cells in models of post-ischaemic brain damage.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA