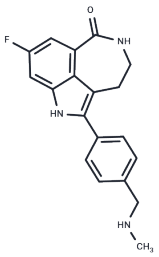
Data Sheet (Cat.No.T4463)

TargetMol

Rucaparib

Chemical Properties



CAS No. : 283173-50-2

Formula: C19H18FN3O

Molecular Weight: 323.36

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	Rucaparib (PF-01367338) is a PARP protein inhibitor (PARP-1 Ki=1.4 nM) and hexose hexose-6-phosphate dehydrogenase (H6PD) inhibitor with oral activity. Rucaparib exhibits antitumor activity, with activity against desmoplasia-resistant prostate cancer (CRPC).
Targets(IC50)	PARP
In vitro	**METHODS**: Glioblastoma cell lines U251 and U87MG were treated with Rucaparib (0.2-100 μM) for 92 h. Cell viability was measured by MTT assay. **RESULTS**: Rucaparib inhibited the proliferation of GBM U251 and U87MG cancer cells in a dose-dependent manner with IC50 values of 14.36 μM and 15.00 μM, respectively.[1] **METHODS**: Human neuroblastoma cells SK-N-BE(2c) and human glioblastoma cells UVW/NAT were treated with Rucaparib (1-10 μM) for 1.5 h and then with H2O2 (20 mM) for 20 min, and the activity of PARP-1 was measured by Immunofluorescence. **RESULTS**: Rucaparib induced a 50% decrease in endogenous PARP-1 activity. treatment with the DNA damaging agent H2O2 resulted in a significant increase in PARP-1 activity, and the H2O2-induced increase in PARP-1 activity was reduced to a level comparable to that of the untreated cells after treatment with Rucaparib. [2]
In vivo	**METHODS**: To detect anti-tumor activity in vivo, BALB/C nude mice bearing U87MG xenografts were treated with Rucaparib (4 mg/kg, intraperitoneally) and BKM120 (15 mg/kg, orally) once daily for 16 days. **RESULTS**: Treatment with BKM120 or Rucaparib alone significantly inhibited tumor growth in terms of volume and weight. When used in combination, tumor growth was further inhibited compared to each drug alone. At the end of the 16-day treatment period, the combination therapy reduced tumor volume and tumor weight by more than 90%. [1]
Kinase Assay	Inhibition of PARP activity in 5×103 D283Med cells is measured using various concentrations of Rucaparib (0-1 μM), compared with DMSO-only. Maximally stimulated PARP activity is measured in samples of permeabilised cells by immunologica.
Cell Research	Medulloblastoma cell lines are seeded in 96-well plates at a density of 1×103, 3×103 and 3×103, respectively. At 24 hours (D384Med) or 48 hours (D283Med and D425Med) after seeding, the cells are exposed to various concentrations of temozolomide in the presence or absence of 0.4 μM Rucaparib. After 3 days (D425Med and D384Med) or 5 days (D283Med) of culture, cell viability is evaluated by a XTT cell proliferation kit assay. Cell growth is expressed as a percentage in relation to DMSO or 0.4 μM Rucaparib-alone

Cell Research	controls. The concentration of temozolomide, alone or in combination with Rucaparib that inhibited growth by 50% (GI50) is calculated. The potentiation factor 50 (PF50) is defined as the ratio of the GI50 of temozolomide in the presence of Rucaparib to the GI50 of temozolomide alone.
Animal Research	Rucaparib is formulated in saline.A single dose of temozolomide is administrated p.o. as a suspension in saline at 200 mg/kg either alone or in combination with a single i.p. administration of PARP inhibitor administered at 0.1 [Rucaparib and MS-AG14644 (equivalent to 0.078 mg/kg free AG14644 only)], 1.0, and 10 mg/kg (for the mesylate salts equivalent to 0.79 and 7.9 mg/kg free AG14451 and AG14452 and 0.78 and 7.8 free AG14531 and AG14644). Control animals are treated with either normal saline p.o. and i. p or normal saline p.o and PARP inhibitor 10 mg/kg i.p.

Solubility Information

Solubility	DMSO: 22.73 mg/mL (70.29 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: 2.27 mg/mL (7.02 mM),Solution. 10% DMSO+90% Saline: < 2.27 mg/mL (7.02 mM),Lower concentrations may be soluble, but exact solubility limit is unknown. *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	3.0925 mL	15.4626 mL	30.9253 mL
5 mM	0.6185 mL	3.0925 mL	6.1851 mL
10 mM	0.3093 mL	1.5463 mL	3.0925 mL
50 mM	0.0619 mL	0.3093 mL	0.6185 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

Zhang S, et al. BKM120 sensitizes glioblastoma to the PARP inhibitor rucaparib by suppressing homologous recombination repair. Cell Death Dis. 2021 May 26;12(6):546.
Lodovichi S, Nepomuceno T C, Woods N T, et al.SART1 modulates Poly-(ADP-Ribose) chain accumulation and PARP1 chromatin localization.iScience.2024
Nile DL, et al. An evaluation in vitro of PARP-1 inhibitors, rucaparib and olaparib, as radiosensitisers for the treatment of neuroblastoma. BMC Cancer. 2016 Aug 11;16:621.
Daniel RA,etal.Central nervous system penetration and enhancement of temozolomide activity in childhood medulloblastoma models by poly(ADP-ribose) polymerase inhibitor AG-014699.Br J Cancer. 2010 Nov 9;103(10):1588-96.
Daniel RA,etal.Inhibition of poly(ADP-ribose) polymerase-1 enhances temozolomide and topotecan activity against childhood neuroblastoma.Clin Cancer Res. 2009 Feb 15;15(4):1241-9.