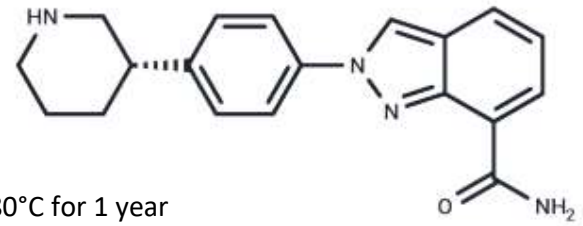
Niraparib [1038915-60-4]

#Cat: NB-64-20714-1ml **Size: 1ml**
#Cat: NB-64-20714-5mg **Size: 5mg**
#Cat: NB-64-20714-50mg **Size: 50mg**
#Cat: NB-64-20714-10mg **Size: 10mg**
#Cat: NB-64-20714-25mg **Size: 25mg**
#Cat: NB-64-20714-200mg **Size: 200mg**
#Cat: NB-64-20714-100mg **Size: 100mg**
#Cat: NB-64-20714-500mg **Size: 500mg**

Chemical Properties



Cas No: 1038915-60-4
Formula: $C_{19}H_{20}N_4O$
Molecular weight: 320.39
Appearance: no data available
Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Niraparib (MK-4827) is a PARP inhibitor that selectively targets PARP1 and PARP2 (IC50=3.8/2.1 nM), exhibiting antitumor activity, inhibiting DNA damage repair, and inducing apoptosis.
Targets(IC50)	Apoptosis,PARP
In vitro	METHODS: The PDAC cell lines MIA-PaCa-2, PANC-1, Capan-1 and the OvCa cell lines OVCAR8, PEO1 were treated with Niraparib (0.1-200 µM) for 48 h, and the cells were assayed for viability using the CellTiter-Glo Luminescent Cell Viability Assay. RESULTS: The IC50 of Niraparib on MIA-PaCa-2, PANC-1, Capan-1, OVCAR8, and PEO1 cells were 26 µm, 50 µm, 15 µM, 20 µM, and 28 µM, respectively.[1] [2]. METHODS: Ovarian cancer cells SKOV3 and UWB1.289 were treated with Niraparib (0.5-15 µM) for 48 h, and the expression levels of target proteins were detected by Western Blot. RESULTS: Niraparib upregulated the expression of PD-L1 in SKOV3 and UWB1.289 cells. [2]
In vivo	**Methods:** To detect anti-tumor activity in vivo, Niraparib (25 mg/kg, administered orally four times per week) and PD-L1 (10 mg/kg, administered intraperitoneally twice per week) were administered to C57BL/6 mice bearing ovarian cancer tumor ID8 for eight weeks. **Results:** Niraparib upregulated PD-L1 expression in ovarian tumors in vivo and synergized with PD-L1 blockade. [2] **Methods:** To detect anti-tumor activity in vivo, Niraparib (50 mg/kg, 0.5% methylcellulose) was administered by gavage to C57BL/6 mice bearing intracranial human-derived TNBC cell lines SUM149, MDA-MB231Br, or MDA-MB-436 once daily for two weeks. **Results:** In the BRCA mutant MDA-MB436 model, Niraparib increased median survival and decreased tumor load. However, it did not increase in the BRCA mutant SUM149 or BRCA wild-type MDA-MB-231Br models, despite high concentrations in intracranial tumors. [3]
Kinase Assay	Enzyme assay is conducted in buffer containing 25 mM Tris, pH 8.0, 1 mM DTT, 1 mM spermine, 50 mM KCl, 0.01% Nonidet P-40, and 1 mM MgCl2. PARP reaction contains 0.1 µCi [3H]NAD+ (200?000 DPM), 1.5 µM NAD+, 150 nM biotinylated NAD+, 1 µg/mL activated calf thymus, and 1?5 nM PARP-1. Autoreactions utilizing SPA bead-based detection are carried out in 50 µL volumes in white 96-well plates. Compounds (e.g., MK-4827) are prepared in 11-point serial dilution in 96-well plate, 5 µL/well in 5%

	DMSO/Water (10× concentrated). Reactions are initiated by adding first 35 µL of PARP-1 enzyme in buffer and incubating for 5 min at room temperature and then 10 µL of NAD+ and DNA substrate mixture. After 3 h at room temperature, these reactions are terminated by the addition of 50 µL of streptavidin-SPA beads (2.5 mg/mL in 200 mM EDTA, pH 8). After 5 min, they are counted using a TopCount microplate scintillation counter. IC50 data is determined from inhibition curves at various substrate concentrations [1].
Cell Research	Proliferation assays were conducted in 96-well black viewplates, and 300 cells/well (250 cell/well for BRCA-1 wt) in culture medium, 190 µL/well (DMEM containing 10% FCS, 0.1 mg/mL penicillin-streptomycin, and 2 mM L-glutamine), were plated and incubated for 4 h at 37℃ under 5% CO2 atmosphere. Inhibitors were then added with serial dilutions, 10 µL/well to obtain the desired final compound concentration in 0.5% DMSO. The cells were then incubated for 7 days at 37℃ in 5% CO2 after which time viability was assessed. Briefly, with CellTiter-Blue solution prediluted 1:10 in medium, 100 µL/well was added and the cells left for 45 min at 37℃ under 5% CO2 and then a further 15 min at room temperature in the dark. The number of living cells was determined by reading the plate at fluorimeter, excitation at 550 nm and emission at 590 nm. Cell growth was expressed as the percentage growth with respect to vehicle treated cells. The concentration required to inhibit cell growth by 50% (CC50) was determined. (Only for Reference)

Solubility Information

Solubility	Ethanol: 60 mg/mL (187.3 mM), H_2O: 1 mg/mL (insoluble or slightly soluble), 10% DMSO+40% PEG300+5% Tween 80+45% Saline: 6 mg/mL (18.73 mM), In vivo: Please add co-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. DMSO: 16.67 mg/mL (52.02 mM), Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.1212 mL	15.606 mL	31.212 mL
5 mM	0.6242 mL	3.1212 mL	6.2424 mL
10 mM	0.3121 mL	1.5606 mL	3.1212 mL
50 mM	0.0624 mL	0.3121 mL	0.6242 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.

Reference

Mobet Y, Wang H, Wei Q, et al. AKAP8 promotes ovarian cancer progression and antagonizes PARP inhibitor sensitivity through regulating hnRNPUL1 transcription. iScience . 2024