## Product Datasheet

# Veliparib dihydrochloride

Catalog No. **BP-02100A** · CAS **912445-05-7** · Form **salt · dihydrochloride** · Physical state **other** · Version **1.0** · Revision **2026-09-14**

**FOR RESEARCH USE ONLY. NOT FOR HUMAN OR VETERINARY USE.**

| Target | Research area |
|---|---|
| **PARP; Autophagy** | **Cancer** |

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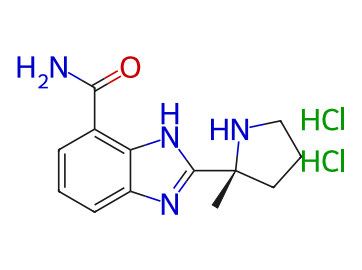
|---|---|
| Molecular formula | **$C_{13}H_{18}Cl_2N_4O$** |
| Molecular weight | **317.2140** |

**STRUCTURE**




## DESCRIPTION

Veliparib (dihydrochloride) is a potent inhibitor of PARP1 and PARP2 with Kis of 5.2 nM and 2.9 nM in cell-free assays, respectively.

## BIOLOGICAL ACTIVITY

In Vitro

Veliparib is inactive to SIRT2 (>5 μM)[1]. Veliparib inhibits the PARP activity with EC50 of 2 nM in C41 cells[2]. Veliparib can decrease the PAR levels in both irradiated and nonirradiated H460 cells. Veliparib reduces clonogenic survival and inhibits DNA repair by PARP-1 inhibition in H460 cells. Veliparib increases apoptosis and autophagy in H460 cells when combination with radiation[3]. Veliparib inhibits PARP activity in H1299, DU145 and 22RV1 cells and the inhibition is independent of p53 function. Veliparib (10 μM) suppresses the surviving fraction (SF) by 43% in the clonogenic H1299 cells. Veliparib shows effective radiosensitivity in oxic H1299 cells. Veliparib can attenuate the SF of hypoxic-irradiated cells including H1299, DU145 and 22RV1.

In Vivo

The oral bioavailability of Veliparib is 56%-92% in mice, SD rats, beagle dogs, and cynomolgus monkeys after oral administration[1]. Veliparib (25 mg/kg, i.p.) can improve tumor growth delay in a NCI-H460 xenograft model. Combination with radiation, veliparib decreases the tumor vessel formation[3]. Veliparib reduces intratumor PAR levels by more than 95% at a dose of 3 and 12.5 mg/kg in A375 and Colo829 xenograft models and the suppression can be maintained over time.

## SOLUBILITY

Soluble in water,DMSO

| | |
|---|---|
| **In vitro** | H2O : 250 mg/mL DMSO : ≥ 3.2 mg/mL |

## REFERENCES

[1]. Donawho CK, et al. ABT-888, an orally active poly(ADP-ribose) polymerase inhibitor that potentiates DNA-damaging agents in preclinical tumor models. Clin Cancer Res. 2007 May 1;13(9):2728-37.

[2]. Penning TD, et al. Discovery of the Poly(ADP-ribose) polymerase (PARP) inhibitor 2-[(R)-2-methylpyrrolidin-2-yl]-1H-benzimidazole-4-carboxamide (ABT-888) for the treatment of cancer. J Med Chem. 2009 Jan 22;52(2):514-23.

[3]. Albert JM, et al. Inhibition of poly(ADP-ribose) polymerase enhances cell death and improves tumor growth delay in irradiated lung cancer models. Clin Cancer Res. 2007 May 15;13(10):3033-42.

[4]. Robert J. Kinders, et al. Preclinical Modeling of a Phase 0 Clinical Trial: Qualification of a Pharmacodynamic Assay of Poly (ADP-Ribose) Polymerase in Tumor Biopsies of Mouse Xenografts. Clin Cancer Res. Author manuscript; available in PMC 2009 Nov 1.

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