Web: www.taiclone.com
Tel: +886-2-2735-9682
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Veliparib

Catalog No: tcsc0076

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Specifications

CAS No:
912444-00-9

Formula:
$C_{13}H_{16}N_4O$

Pathway:
Epigenetics;Cell Cycle/DNA Damage;Autophagy

Target:
PARP;PARP;Autophagy

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 29 mg/mL (118.71 mM)

Alternative Names:
ABT-888

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Observed Molecular Weight:
244.29

Product Description

Veliparib is a potent **PARP** inhibitor, inhibiting **PARP1** and **PARP2** with **K_i**s of 5.2 and 2.9 nM, respectively.

IC50 & Target: Ki: 5.2 nM (PARP1), 2.9 nM (PARP2)[1]

In Vitro: Veliparib (ABT-888) is also tested against SIRT2, an enzyme that also uses NAD^+ for catalysis, and found to be inactive (>5,000 nM). The receptor profile of Veliparib is determined in a panel of 74 receptor-binding assays at a concentration of 10 μM. Veliparib displaces control-specific binding at 50% or greater at the human H_1(61%), the human 5-HT_{1A} (91%), and the human 5-HT_7 (84%) sites only. The IC_{50}s for these three receptors are 5.3, 1.5, and 1.2 μM, respectively[1]. c-Met knockdown cells show 4.2- (shMet-A; 95% CI=4-4.5) or 4.6-fold (shMet-B; 95% CI=4.4-4.8) growth inhibition when treated with 60 μM Veliparib (ABT-888). When treated with 38 μM Veliparib, c-Met knockdown cells show 2- (shMet-A; 95% CI=1.5-2.5) or 1.9-fold (shMet-B; 95% CI=1.3-2.5) growth inhibition[2]. In HaCaT cells, at 6 h post-treatment by Veliparib (ABT-888), cell viability is significantly increases under 1,000 μM sulfur mustard (SM) exposure, whereas Veliparib does not protect cell viability under 100 μM SM exposure. Moreover, the addition of Veliparib no longer shows the protective effect at 24 h post SM exposure[3].

In Vivo: Veliparib (ABT-888) is a potent inhibitor of PARP, has good oral bioavailability, can cross the blood-brain barrier, and potentiates temozolomide, platinums, cyclophosphamide, and radiation in syngeneic and xenograft tumor models[1]. In MDA-MB-231 xenograft tumor models, combination treatment (AG014699/Crizotinib and Veliparib (ABT-888)/Foretinib) substantially reduced tumor growth compared to either inhibitor alone[2].