# bpV(phen) trihydrate

**Cat. No.:** HY-122818

**CAS No.:** 171202-16-7

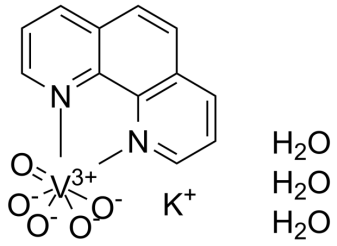
**Molecular Formula:** $C_{12}H_{14}KN_2O_8V$

**Molecular Weight:** 404.29

**Target:** PTEN; Phosphatase; Parasite; Apoptosis

**Pathway:** PI3K/Akt/mTOR; Metabolic Enzyme/Protease; Anti-infection; Apoptosis

**Storage:** 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)




## SOLVENT & SOLUBILITY

**In Vitro**

$H_2O$ : 18.18 mg/mL (44.97 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 2.4735 mL | 12.3674 mL | 24.7347 mL |
| | 5 mM | 0.4947 mL | 2.4735 mL | 4.9469 mL |
| | 10 mM | 0.2473 mL | 1.2367 mL | 2.4735 mL |

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

**In Vivo**

1. Add each solvent one by one: PBS
Solubility: 25 mg/mL (61.84 mM); Clear solution; Need ultrasonic and warming

## BIOLOGICAL ACTIVITY

**Description**

bpV(phen) trihydrate, a insulin-mimetic agent, is a potent protein tyrosine phosphatase (PTP) and PTEN inhibitor with $IC_{50}$s of 38 nM, 343 nM and 920 nM for PTEN, PTP-β and PTP-1B, respectively. bpV(phen) trihydrate inhibits proliferation of the protozoan parasite ***Leishmania*** in vitro. bpV(phen) trihydrate strongly induces the secretion of a large number of chemokines and pro-inflammatory cytokines, and it activates a Th1-type pathway (IL-12, IFNγ). bpV(phen) trihydrate can also induce cell apoptosis, and has anti-angiogenic and anti-tumor activity[1][2][3][4][5].

**$IC_{50}$ & Target**

IC50: 38 nM (PTEN), 343 nM (PTP-β) and 920 nM (PTP-1B)[3]
Parasite Leishmania[2]
Apoptosis[1]

**In Vitro**

bpV(phen) (5 μM; 24.5 hours; H9c2 cells) treatment causes a further decrease of cell viability in H/R-injured H9c2 cells[1].
bpV(phen) (5 μM; 24.5 hours; H9c2 cells) treatment increases the apoptosis of H/R-injured H9c2 cells[1].
bpV(phen) (5 μM; 24.5 hours; H9c2 cells) treatment significantly promotes the accumulation of cytoplasmic Cytochrome C in H/R-injured H9c2 cells[1].

After stimulation of bpV(phen), PTEN-induced putative kinase protein 1 (PINK1)/Parkin-mediated mitophagy is inhibited[1]. bpV(phen) is an insulin-mimetic agent following insulin-receptor tyrosine kinase hyperphosphorylation and activation[4].

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

Cell Viability Assay[1]

| | |
|---|---|
| Cell Line: | Hypoxia/reoxygenation (H/R)-injured H9c2 cells |
| Concentration: | 5 μM |
| Incubation Time: | 24.5 hours (hypoxia for 24 h; reoxygenation for 30 minutes) |
| Result: | Caused a further decrease of cell viability. |

Apoptosis Analysis[1]

| | |
|---|---|
| Cell Line: | Hypoxia/reoxygenation (H/R)-injured H9c2 cells |
| Concentration: | 5 μM |
| Incubation Time: | 24.5 hours (hypoxia for 24 h; reoxygenation for 30 minutes) |
| Result: | Increased the apoptosis of H/R-injured H9c2 cells. |

Western Blot Analysis[1]

| | |
|---|---|
| Cell Line: | Hypoxia/reoxygenation (H/R)-injured H9c2 cells |
| Concentration: | 5 μM |
| Incubation Time: | 24.5 hours (hypoxia for 24 h; reoxygenation for 30 minutes) |
| Result: | Showed an increased release of Cytochrome C. |

**In Vivo**

bpV(phen) (5 mg/kg; intraperitoneal injection; daily; for 38 days; male BALB/c nude (nu/nu) athymic mice) treatment causes a significant reduction in average tumor volume[1].

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

| | |
|---|---|
| Animal Model: | Male BALB/c nude (nu/nu) athymic mice (6-7 weeks old) injected with PC-3 cells[2] |
| Dosage: | 5 mg/kg |
| Administration: | Intraperitoneal injection; daily; for 38 days |
| Result: | Caused a significant reduction in average tumor volume. |

## CUSTOMER VALIDATION

- Nat Commun. 2022 May 19;13(1):2762.
- Biochem Bioph Res Co. 2020 Sep 3;529(4):1045-1052.

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## REFERENCES

[1]. Tang W, et al. PTEN-mediated mitophagy and APE1 overexpression protects against cardiac hypoxia/reoxygenation injury. In Vitro Cell Dev Biol Anim. 2019 Oct;55(9):741-748.

[2]. Caron D, et al. Protein tyrosine phosphatase inhibition induces anti-tumor activity: evidence of Cdk2/p27 kip1 and Cdk2/SHP-1 complex formation in human ovarian cancer cells. Cancer Lett. 2008 Apr 18;262(2):265-75.

[3]. Schmid AC, et al. Bisperoxovanadium compounds are potent PTEN inhibitors. FEBS Lett. 2004 May 21;566(1-3):35-8.

[4]. Band CJ, et al. Early signaling events triggered by peroxovanadium [bpV(phen)] are insulin receptor kinase (IRK)-dependent: specificity of inhibition of IRK-associated protein tyrosine phosphatase(s) by bpV(phen). Mol Endocrinol. 1997 Dec;11(13):1899-910.

[5]. Chen Q, et al. Potassium Bisperoxo(1,10-phenanthroline)oxovanadate (bpV(phen)) Induces Apoptosis and Pyroptosis and Disrupts the P62-HDAC6 Protein Interaction to Suppress the Acetylated Microtubule-dependent Degradation of Autophagosomes. J Biol Chem. 201



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