bpV(phen)

®

MedChemExpress

Cat. No.:	HY-136065	
CAS No.:	42494-73-5	
Molecular Formula:	C ₁₂ H ₈ KN ₂ O ₅ V	Ļ
Molecular Weight:	350.24	
Target:	PTEN; Phosphatase; Parasite; Apoptosis	_
Pathway:	PI3K/Akt/mTOR; Metabolic Enzyme/Protease; Anti-infection; Apoptosis	C
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.	0

 K^+

Description	bpV(phen), a insulin-mime 343 nM and 920 nM for PT <i>Leishmania</i> in vitro. bpV(p cytokines, and it activates angiogenic and anti-tumo	etic agent, is a potent protein tyrosine phosphatase (PTP) and PTEN inhibitor with IC ₅₀ s of 38 nM, EN, PTP-β and PTP-1B, respectively. bpV(phen) inhibits proliferation of the protozoan parasite ohen) strongly induces the secretion of a large number of chemokines and pro-inflammatory s a Th1-type pathway (IL-12, IFNγ). bpV(phen) can also induce cell apoptosis, and has anti- or activity ^{[1][2][3][4][5]} .
IC ₅₀ & Target	IC50: 38 nM (PTEN), 343 nI Parasite Leishmania ^[2] Apoptosis ^[1]	M (PTP-β) and 920 nM (PTP-1B) ^[3]
In Vitro	bpV(phen) (5 μM; 24.5 hou bpV(phen) (5 μM; 24.5 hou bpV(phen) (5 μM; 24.5 hou H/R-injured H9c2 cells ^[1] . After stimulation of bpV(p bpV(phen) is an insulin-m MCE has not independent Cell Viability Assay ^[1]	urs; H9c2 cells) treatment causes a further decrease of cell viability in H/R-injured H9c2 cells ^[1] . urs; H9c2 cells) treatment increases the apoptosis of H/R-injured H9c2 cells ^[1] . urs; H9c2 cells) treatment significantly promotes the accumulation of cytoplasmic Cytochrome C in when), PTEN-induced putative kinase protein 1 (PINK1)/Parkin-mediated mitophagy is inhibited ^[1] . imetic agent following insulin-receptor tyrosine kinase hyperphosphorylation and activation ^[4] . cly confirmed the accuracy of these methods. They are for reference only.
	Cell Line:	Hypoxia/reoxygenation (H/R)-injured H9c2 cells
	Concentration:	5 μΜ
	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 μΜ
	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 μΜ
	Incubation Time:	24.5 hours (hypoxia for 24 h; reoxygenation for 30 minutes)
	Result:	Showed an increased release of Cytochrome C.
ı Vivo	bpV(phen) (5 mg/kg; int a significant reduction in MCE has not independe	rraperitoneal injection; daily; for 38 days; male BALB/c nude (nu/nu) athymic mice) treatment caus n average tumor volume ^[1] . ently confirmed the accuracy of these methods. They are for reference only.
ı Vivo	bpV(phen) (5 mg/kg; int a significant reduction in MCE has not independe Animal Model:	raperitoneal injection; daily; for 38 days; male BALB/c nude (nu/nu) athymic mice) treatment caus n average tumor volume ^[1] . Intly confirmed the accuracy of these methods. They are for reference only. Male BALB/c nude (nu/nu) athymic mice (6-7 weeks old) injected with PC-3 cells ^[2]
ı Vivo	bpV(phen) (5 mg/kg; int a significant reduction in MCE has not independe Animal Model: Dosage:	raperitoneal injection; daily; for 38 days; male BALB/c nude (nu/nu) athymic mice) treatment cau n average tumor volume ^[1] . ently confirmed the accuracy of these methods. They are for reference only. Male BALB/c nude (nu/nu) athymic mice (6-7 weeks old) injected with PC-3 cells ^[2] 5 mg/kg
ı Vivo	bpV(phen) (5 mg/kg; int a significant reduction in MCE has not independe Animal Model: Dosage: Administration:	erraperitoneal injection; daily; for 38 days; male BALB/c nude (nu/nu) athymic mice) treatment cau n average tumor volume ^[1] . ently confirmed the accuracy of these methods. They are for reference only. Male BALB/c nude (nu/nu) athymic mice (6-7 weeks old) injected with PC-3 cells ^[2] 5 mg/kg Intraperitoneal injection; daily; for 38 days

CUSTOMER VALIDATION

• Biochem Bioph Res Co. 2020 Sep 3;529(4):1045-1052.

See more customer validations on www.MedChemExpress.com

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. 2015 Oct 23;290(43):26051-8.

Caution: Product has not been fully validated for medical applications. For research use only.

 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