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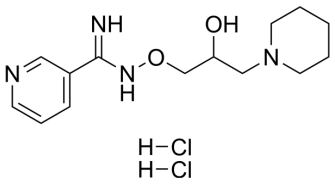
BGP-15

Cat. No.:	HY-100828
CAS No.:	66611-37-8
Molecular Formula:	$C_{14}H_{24}Cl_2N_4O_2$
Molecular Weight:	351.27
Target:	PARP
Pathway:	Cell Cycle/DNA Damage; Epigenetics
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 : 100 mg/mL (284.68 mM; Need ultrasonic)
DMSO : 11.33 mg/mL (32.25 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8468 mL	14.2341 mL	28.4681 mL
5 mM	0.5694 mL	2.8468 mL	5.6936 mL
10 mM	0.2847 mL	1.4234 mL	2.8468 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (284.68 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.12 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.12 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.12 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	BGP-15 is a PARP inhibitor, with an IC_{50} and a K_i of 120 and 57 μM, respectively.
IC_{50} & Target	PARP 120 μM (IC_{50})
In Vitro	BGP-15 (200 μM) prevents the imatinib mesylate-induced oxidative damages, attenuates the depletion of high-energy

	phosphates, alters the signaling effect of imatinib mesylate by preventing p38 MAP kinase and JNK activation, and induced the phosphorylation of Akt and GSK-3beta[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BGP-15 (15 mg/kg, p.o.) does not improve skeletal muscle pathology in older mdx mice[1]. In a rat model, 10 days of BGP-15 treatment greatly improves diaphragm muscle fiber function (by about 100%), although it does not reverse diaphragm atrophy. The treatment also provides protection from myosin PTMs associated with HSP72 induction and PARP-1 inhibition, resulting in improvement of mitochondrial function and content[2]. BGP-15 (15 mg/kg per day in saline) treatment has no effect in Ntg mice or an independent cohort of normal adult wild-type mice based on morphology, cardiac function and ECG parameters. Treatment with BGP-15 attenuates the increase in atrial size and lung weight. BGP-15 treatment is able to prevent or reduce episodes of arrhythmia. BGP-15 treatment is associated with a reduced PR interval in the HF+AF model[3]. BGP-15 (10 and 30 mg/kg) increases insulin sensitivity by 50% and 70%, respectively, in cholesterol-fed but not in normal rabbits. After 5 days of treatment with BGP-15, the glucose infusion rate is increased in a dose-dependent manner in genetically insulin-resistant GK rats. The most effective dose is 20 mg/kg, which shows a 71% increase in insulin sensitivity compared to control group[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3]	Adult (appr 4 month) male HF+AF and Ntg mice are administered with BGP-15 (15 mg/kg per day in saline) for 4 weeks by oral gavage or remained untreated (oral gavage with saline or no gavage). Gavage with saline has no effect on morphological or functional parameters in the HF+AF model. Therefore, untreated mice (no gavage) and mice administered saline are combined and referred to as HF+AF control. Echocardiography and ECG studies are performed before and after treatment. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Redox Biol. 2023 Apr 6,102697.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

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[2]. Salah H, et al. The chaperone co-inducer BGP-15 alleviates ventilation-induced diaphragm dysfunction. Sci Transl Med. 2016 Aug 3;8(350):350ra10

[3]. Sapra G, et al. The small-molecule BGP-15 protects against heart failure and atrial fibrillation in mice. Nat Commun. 2014 Dec 9;5:5705

[4]. Literati-Nagy B, et al. Improvement of insulin sensitivity by a novel drug candidate, BGP-15, in different animal studies. Metab Syndr Relat Disord. 2014 Mar;12(2):125-31

[5]. Sarszegi Z, et al. BGP-15, a PARP-inhibitor, prevents imatinib-induced cardiotoxicity by activating Akt and suppressing JNK and p38 MAP kinases. Mol Cell Biochem. 2012 Jun;365(1-2):129-37

[6]. Szabados E, et al. BGP-15, a nicotinic amidoxime derivate protecting heart from ischemia reperfusion injury through modulation of poly(ADP-ribose) polymerase.

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