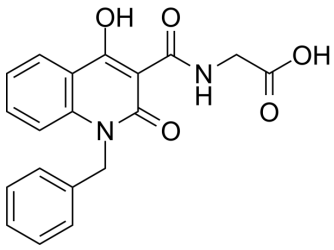
IOX2

Cat. No.:	HY-15468
CAS No.:	931398-72-0
Molecular Formula:	$C_{19}H_{16}N_2O_5$
Molecular Weight:	352.34
Target:	HIF/HIF Prolyl-Hydroxylase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (70.95 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8382 mL	14.1908 mL	28.3817 mL
	5 mM	0.5676 mL	2.8382 mL	5.6763 mL
	10 mM	0.2838 mL	1.4191 mL	2.8382 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

IOX2 is a specific prolyl hydroxylase-2 (PHD2) inhibitor with IC_{50} of 22 nM. IOX2 regulates platelet function and arterial thrombosis by upregulating HIF-1α expression and inhibiting ROS production. IOX2 can be used in the study of thrombotic diseases[1][2].

IC_{50} & Target

IC50: 22 nM (PHD2)[2]

In Vitro

IOX2 (0, 10, 25, and 50 μM) dose-dependently inhibits collagen-related peptide (CRP; 0.25 μg/mL) or thrombin (0.03 U/mL)-induced platelet aggregation and ATP release. But IOX2 doesn't affect P-selectin expression and the surface levels of glycoprotein (GP)Ibα, GPVI, or αIIbβ3[1].

IOX2 also inhibits the spreading of platelets on fibrinogen or collagen and clot retraction[1].

IOX2 (50 μM; 24 h) increases the transcription level of VEGF-A and BNIP3 in Normal human epidermal keratinocytes (NHEK) and Normal human dermal fibroblasts (NHDF), when grown under normoxia and hypoxia condition[2].

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

In Vivo

IOX2 (10 mg/kg; i.p.; single dose) impaired the in vivo hemostatic function of platelets and arterial thrombus formation in mice[1].

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Animal Model:	Mouse[1]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection
Result:	Upregulated HIF-1α in platelets, decreased ROS generation, and downregulated NOX1 expression. Increased the phosphorylation level of VASP (Ser157/239), and inhibited the phosphorylation of p38 (Thr180/Tyr182), ERK1/2 (Thr202/Tyr204), AKT (Thr308/Ser473), and PKCδ (Thr505) in CRP- or thrombin-stimulated platelets.

CUSTOMER VALIDATION

- Nat Commun. 2022 May 4;13(1):2447.
- Thromb Haemostasis. 2022 Apr 27.
- Aging. 2021 May 20;13(10):14355-14371.
- Sci Rep. 2022 Jan 27;12(1):1443.
- Mediat Inflamm. 05 Aug 2021.

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REFERENCES

[1]. Gu W, et al. Inhibition of Hypoxia-Inducible Factor Prolyl-Hydroxylase Modulates Platelet Function. Thromb Haemost. 2022 Oct;122(10):1693-1705.

[2]. Deppe J, et al. Impairment of hypoxia-induced HIF-1α signaling in keratinocytes and fibroblasts by sulfur mustard is counteracted by a selective PHD-2 inhibitor. Arch Toxicol. 2016 May;90(5):1141-50.

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